

UNIVERSITÄT PADERBORN  
SPORTMEDIZINISCHES INSTITUT

**SENSORIMOTOR CONTROL AND ASSOCIATED  
BRAIN ACTIVITY IN SPORTS MEDICINE  
RESEARCH**

Kumulative Habilitation  
zur Erlangung der *venia legendi* für das Fach  
*Sportmedizin*  
an der Universität Paderborn

vorgelegt im März 2013 von

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## Abbreviations

ACC	Anterior Cingulate Cortex
ACL	Anterior Cruciate Ligament
BA	Brodman Area
CNS	Central Nervous System
DLPFC	Dorsolateral Prefrontal Cortex
EEG	Electroencephalography
EMG	Electromyography
ERP	Event Related Potential
F	Female
FFT	Fast Fourier Transformation
fNIRS	Functional Near-Infrared Spectroscopy
fMRI	Functional Magnetic resonance imaging
GTO	Golgi-Tendon-Organ
Hz	Hertz
ICA	Independent Component Analysis
M	Male
MT	Motor Time
MVC	Maximal Voluntary Contraction
NIRS	Near-Infrared Spectroscopy
OFC	Orbitofrontal Cortex
PET	Positron Emission Tomography

PMC	Premotor Cortex
PNS	Peripheral Nervous System
RT	Reaction Time
SMA	Supplementary Motor Area
VO <sub>2max</sub>	Maximal Oxygen Consumption
YRS	Years

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Above all, my deepest thanks go to my family Kornelia, Luca and Neele. I know life would be nothing without you.

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<sup>2</sup> Only figures excluding the the figures from the publications 1 to 10 will be displayed



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## List of Publications included in the cumulative habilitation thesis

1. Baumeister, J., Reinecke, K., Schubert, M., Schade, J., & Weiss, M. (2012). Effects of induced fatigue on brain activity during sensorimotor control. *Eur J Appl Physiol* 112(7) 2475-82
2. Baumeister, J., von Detten, S., van Niekerk, S.M., Schubert, M., Ageberg, E. & Louw, Q.A. (2013). Brain activity in predictive sensorimotor control for landings: an EEG pilot study. *Int J Sports Med* (accepted for publication on 21.2.2013)
3. Baumeister, J., Barthel, T., Geiss, K. R., & Weiss, M. (2008). Influence of phosphatidylserine on cognitive performance and cortical activity after induced stress. *Nutr Neurosci* 11(3), 103–110
4. Baumeister, J., Reinecke, K., Liesen, H., & Weiss, M. (2008). Cortical activity of skilled performance in a complex sports related motor task. *Eur J Appl Physiol* 104(4), 625–631
5. Baumeister, J., Reinecke, K., Cordes, M., Lerch, C., & Weiss, M. (2010). Brain activity in goal-directed movements in a real compared to a virtual environment using the Nintendo Wii. *Neurosci Lett* 481, 47-50
6. Reinecke, K., Cordes, M., Lerch, C., Koutsandreou, F., Schubert, M., Weiss, M. & Baumeister J. (2011). From Lab to Field Conditions: A Pilot Study on EEG Methodology in Applied Sports Science. *Appl Psychophys Biofeedback* 36, 265-271
7. Baumeister, J. (2012). What the brain can tell us in musculoskeletal rehabilitation. *J Sports Med Dopng Stud*, 2(3), 1-2
8. Baumeister, J., Reinecke K. & Weiss M. (2008). Changed cortical activity after anterior cruciate ligament reconstruction in a joint position paradigm: an EEG study. *Scand J Sci Med Sports* 18, 473-484<sup>3</sup>

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<sup>3</sup> This publication contains data from my dissertation thesis. It is used to give a better overview about the effects of the injury-related condition

9. Baumeister, J., Reinecke K., Schubert M. & Weiss M. (2011). Altered electrocortical brain activity after ACL reconstruction during force control. *J Orthop Res* 29(9), 1383-9
10. Plattner, K., Lambert, M.I., Tam, N. & Baumeister, J. (2012). The response of cortical alpha activity to pain and neuromuscular changes caused by exercise induced muscle damage. *Scand J Sci Med Sports* [epub ahead of print]

## CHAPTER 1

### Introduction

Sports Medicine refers to a broad field related to physical activity, exercise and sport and is classified as relating either to performance enhancement or to injury care and management. Sports medicine is based on physiology, the scientific study of function in living biological systems. Up to now, it covers the more established topics of sports and exercise like cardiovascular, energetic, endocrine, pulmonary and neuromuscular function and adaptability. While physiology has done a high amount of research in these topics over the last 50 years, aspects of neurophysiology have been largely ignored. Even though everyone knows that brain function is important to sports and exercise (and to all other biological systems as well), one can only speculate about how the brain drives and adapts to sport and exercise.

How do we control movement? How does the brain regulate its millions of nerve cells to produce motor behavior? The Nobel Prize laureates Charles Scott Sherrington and Eric R. Kandel are two of the founders of modern neuroscience. From a neurophysiological perspective they propose functions of neurons and memory storage in neurons as key areas in motor behavior. Nowadays we try to discover the relationship between motor behavior and activities of the brain. The progress in methodological approaches in neuroscience has given us the possibility to open a window to the brain by using sophisticated methods like Positron-Emission-Tomography (PET), functional Magnet-Resonance-Imaging (fMRI), structural MRI, functional Near-infrared Spectroscopy (fNIRS) Magnetelectroencephalography (MEG) and Electroencephalography (EEG).

The time has come now to introduce these methods also to the field of sports medicine. Therefore, this work will demonstrate an electrophysiological approach to record, detect and interpret changes in brain activity related to sensorimotor control. Chapter 2 will give an

overview about the physiological background and opportunities to measure sensorimotor control according to its components motor behavior, sensory reception and information processing. Chapter 3 will identify the electroencephalography as the method of choice to look into the cortical processing and integration of information, before Chapter 4 will introduce the working-memory-hypothesis into the process of sensorimotor control. To give an example of what is possible, how brain activity can be measured in the light of the working-memory-hypothesis and how this can contribute to sensorimotor control, Chapters 5-8 involve the research program with studies done in stress-related, sports-related and injury related conditions.

It is important to say that we are still at the very beginning to understand the underlying mechanisms and functions of the brain according to sensorimotor control in sports and exercise. This work will contribute to these beginnings and help to open a largely ignored but very important field in sports medicine research. There is still a lot to do, so let's start doing it...

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## CHAPTER 2

### Physiological insights and measures of Sensorimotor Control

Coordinated movements in sports and exercise require multiple, intact muscle innervation in order to move precisely and in an economic way. Imagine the gymnast during his floor exercises, think about the soccer player while passing a ball to a teammate or visualize a golf player performing a difficult putt. In all cases multiple muscles are coordinated without interference. It just looks trivial – but it is not!

The human body is highly maneuverable in a multitude of coordinated movements (Enoka, 2008). The process of motor coordination is defined as interplay of biological systems to organize goal-directed movements (Meinel, Schnabel, & Krug, 2007). The interplay is mediated by sensory information feedback, enabling adjustments to the ever changing environment. Neural representations in the central nervous system (CNS) formed by different sensory modalities are the framework in which movements are planned, accommodated and executed (Amaral, 2013).

The physiological concept behind motor coordination is based on sensory input, processing and motor executive systems. In this context, sensorimotor control is specified as a dynamic interaction between sensations of sensory stimuli, the processing and integration of this information in the CNS and motor behavior in voluntary movement and postural control<sup>4</sup> (Riemann & Lephart, 2002; Baumeister, Reinecke, Schubert, Schade, & Weiss, 2012). From a physiological perspective it includes two biological systems, the musculoskeletal and the nervous system. The human musculoskeletal system includes bones, joints, skeletal muscles, tendons and ligaments and provides mechanical stability and movements of the body. The nervous system is divided into a central (CNS) and peripheral (PNS) part. The

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<sup>4</sup> The sensorimotor control model also provides postural control to the human body, but this topic will be not discussed here. For a review see Jacobs & Horak, 2007

PNS includes the afferent (sensory) and efferent (motor) neurons, whereas the CNS consists of processing centers in the brain and the spinal cord (Silverthorn, 2010).

One of the most popular concepts of human functioning in a real world environment is based on the fundamental notion that humans are processors of information (Schmidt & Lee, 2011). Besides visual and vestibular stimuli, proprioceptive information (representing the modalities of joint position sense, force sensation and the sensation of motion stimuli) are important sensory modalities for sensorimotor control to provide adaptability to the environment. Sensory modalities are processed at the three levels (1) the reception by sense organs, (2) processing by the brain and (3) the motor behavior provided by the peripheral muscles (Figure 2-1).

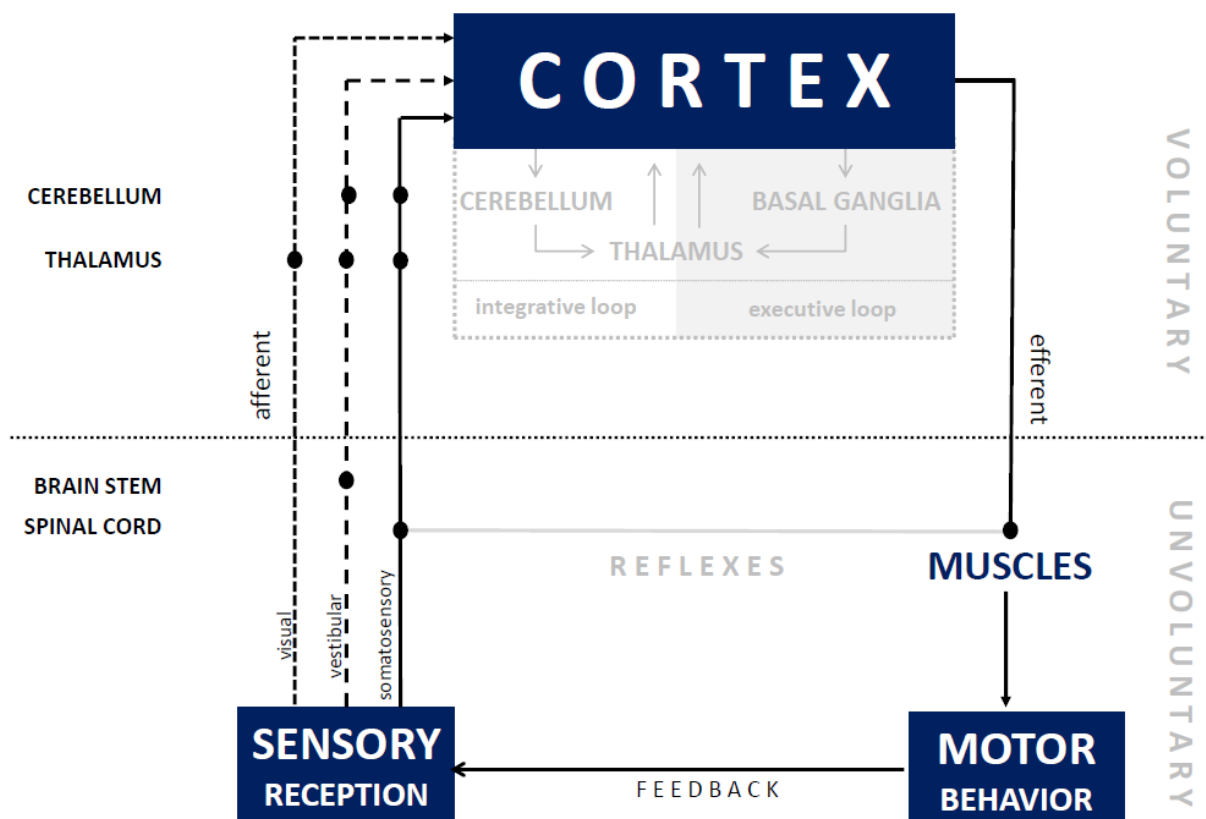


FIGURE 2-1: Model of sensorimotor control and its different levels of information processing

The following sections will give a detailed look into the three levels of sensorimotor control and will provide the background for a rationale for the next step in sports medicine research. By having a closer look into the brain we will be able to detect principles and mechanisms for the understanding of sports and exercise including prevention and rehabilitation.

### **a. Motor Behavior in Sensorimotor Control**

Movement can be specified as “the heart and soul” of sports and exercise. The coordination of movement is accomplished by the interaction of the nervous system with the muscles. The nervous system controls muscle forces via electrophysiological signals from motor neurons to the muscle fibers. The applied muscle force results in coordinated movements. In this efferent branch of sensorimotor control the coupling of the motor neuron with the muscle fibers is known as ‘motor unit’ (Silverthorn, 2010). This basic concept had already been proposed by Sherrington in 1925. Muscle force depends on the characteristics of motor unit activity and on the arrangement of muscle fibers only before these forces provide goal directed motor behavior.

The neuronal activation of the muscle fibers by action potentials can be measured with surface electrodes. The methodology is known as electromyography (EMG; Enoka, 2008). This technique is extensively used and adopted for the field of biomechanics (Luca, 1997), even though it is a physiological measurement. The EMG is able to quantify the magnitude of the amplitude or the frequency spectrum of the electrophysiological signal. It adds important knowledge to the field of sports and exercise in helping to quantify efferent muscle activation within a given time. It is also helpful in identifying muscle activity pattern related to motor behavior in more than one muscle. For a deeper insight, recommendations and limitations of the EMG methodology have been described by (Hermens, 1999) in the SENIAM project in “*European Recommendations for Surface Electromyography*”.

However, coordinated movement is traditionally described with non-physiological variables in sports and exercise, which can be observed from outside the body (known as behavioral

approach in kinesiology, motor control and biomechanics). The analysis of motor behavior from an outside perspective involves the description of the outcomes and characteristics of a movement.

Three fundamental ways characterize the outcome of a movement and assess the achievement of environmental goals: measures of error, speed and movement magnitude. In many cases performances in sports and exercise need to be highly precise and require minimum error. Researchers pinpoint different measures of error<sup>5</sup> to assess the accuracy of motor behavior. Typical tasks for measuring motor behavior are tracking and position or force matching tasks which require an ongoing performance which rely on sensory feedback (Schmidt & Lee, 2011). The measurement of speed is another basic measure of motor behavior. Here, the researcher assumes that a performer who can achieve more in a given time or can produce the same motor behavior in less time is thought to be more skillful. Typical measures of speed and time are reaction times (RT) and movement times (MT). RT measures are conducted extensively in sensorimotor control studies as a measure of sensory information processing speed, whereas MT describes the interval from the initiation of response at the end of RT and last till the execution of the response movement is finished. A third measure which is important, especially in sports and exercise as a determinant of success, is the magnitude of motor behavior. This measure assesses distances or weights and is used only rarely associated to assessments in motor behavior. In conclusion, accuracy, speed and magnitude are able to analyze the achievement of environmental goals and therefore are able to describe the outcome of a movement.

In addition to the motor outcome, biomechanics is able to analyze the characteristics of a movement. The field of biomechanics is defined as “the application of the principles of mechanics to study biological systems” (Schmidt & Lee, 2011; Enoka, 2008). Two branches of biomechanics are described to characterize movements, the kinematic and the kinetic description.

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<sup>5</sup> Schmidt & Lee, 2011 have described computations and interpretation of all different error measures in detail.



The kinematic analysis completely ignores the cause of motion and involves measures of position, displacement, velocity and acceleration. The position of an object, a person's whole body or a segment of the body refers to a location in space associated to some baseline value or axis. When the position is changed, the body or object has been displaced and motion has occurred. Motion can be described as an event in space and time and involves not only the change in position, but also includes the terms velocity, speed and acceleration (Enoka 2008). Modern motion capture systems are based on optical camera systems and are able to analyze changes in these variables with a high accuracy. This technique is often used to evaluate training interventions (e.g. golf Egret, Nicolle, Dujardin, Weber, & Chollet, 2006; Beak et al., 2013) or to distinguish between different subpopulations e.g. according to injuries (Besier, Lloyd, Ackland, & Cochrane, 2001; Waite, Beard, Dodd, Murray, & Gill, 2005) especially in sports medicine. In the kinetic analysis, force is a causal agent that produces or tends to produce a change in the state of rest or motion of a body or object. Force is used to analyze the physical interaction of an object with its surroundings (Enoka, 2008). Forces experienced by the human body during movement can be categorized into linear or angular kinetics (Winter, 2009). Linear kinetics examines the relation between a body's resistance to a change in its linear state of motion, whereas angular kinetics describe the relation between a body's resistance in its angular state of motion. The forces in the human body include joint reaction and muscle forces. In biomechanics, 3D motion capture systems combined with force plates are often used to calculate detailed kinematic and kinetic data and therefore characterize motor behavior in every detail (e.g. Louw, Grimmer, & Vaughan, 2006).

What are the underlying mechanisms behind the scenes of motor behavior? In order to understand movement and therefore sensorimotor control we need to have a deeper look into the neurophysiological processes behind the motor output. Therefore it is important to switch from an 'outside the body perspective' into an 'inside point of view'.

## **b. Sensory Reception in Sensorimotor control**

Sensory systems provide and customize internal representations of the body itself and the external environment. These representations control movement coordination and lead to goal-directed motor behavior. Sensory receptors support the CNS with the information necessary for movement control. The underlying process where information about the internal state and the external surroundings is fed back to the CNS is termed feedback. It is a basic principle in physics/engineering as well as in biological systems that the more maneuverable a system, the more feedback it requires for reaching its goals (Hasan & Stuart, 1988). Movement coordination is highly maneuverable and therefore requires a considerable amount of feedback. This is evident in the number of afferent neurons which is much greater in number in the human body compared to the number of efferent neurons used for activating muscles (Enoka, 2008).

Sensory systems transform physical energy in neural signals through a process termed transduction (Gardner & Johnson, 2013a). Sensory transduction is performed by all sensory systems and involves either a chemical (e.g. retina photo transduction) or mechanical (e.g. in somatosensory systems) process.

The most important sensory information in sensorimotor control besides visual and vestibular information is proprioception. Proprioception is one of the most misused terms in sports and exercise. In 1906 one of the founders of modern neuroscience, the Nobel Prize laureate Charles S. Sherrington originally formed the term proprioception as “the perception of joint and body movement as well as position of the body, or segments of the body, in space” (Sherrington, 1906). Even though Sherrington used perception in his description, nowadays definitions refer only to the afferent sensory information input to the CNS without the transfer into perception (Quante & Hille, 1999). At present, proprioception is specified as the cumulative afferent information arising from internal peripheral areas of the body from specialized nerve endings called mechanoreceptors and contribute to the perception of limb relative position, body/joint movement and tension/force to regulate sensorimotor control

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(Riemann & Lephart, 2002). Within this scope, proprioception is divided into three different modalities: the sensation of (1) motion, (2) joint position and (3) force or tension (Riemann & Lephart, 2002; Baumeister, Reinecke, Schubert, & Weiß, 2011). Proprioceptive information originates from sensory neurons located in the muscle, tendon, fascia, joint capsule, ligament, and skin (Carpenter, Blasler, & Pellizzon, 1998). Although sensation generally occurs in the same manner across various mechanoreceptors, each type of receptor possesses a degree of specificity for the sensory modality for which it responds and the range of stimuli within the modality.

In brief, muscle spindles are encapsulated sense organs and responsible for detecting muscle length and rate of changes in muscle length (Silverthorn, 2010). There are about 27.500 muscle spindles in the human body with hand and neck muscles having the highest density (Prochazka, 1996). Muscle spindles are fusiform shaped and are parallel to skeletal muscle fibers (extrafusal). Within a muscle spindle, there are several small, specialized muscle fibers known as intrafusal fibers. There are two types of intrafusal fibers, the nuclear chain fibers (sensitive to the rate of change in muscle length) and the nuclear bag fibres (sensitive to the absolute muscle length). The center of an intrafusal fibers lack the contractile element myosin, so that contraction only occurs at the ends. The peripheral ends are innervated independent of extrafusal muscle fibers via gamma motor neurons. Activation of peripheral the contractile elements causes stretching/elongation of the central region and results in an increase in firing rate of the sensory endings. This leads to an increase in sensitivity of the muscle spindle to changes in length (Gardner & Johnson, 2013a). In summary, muscle spindles are part of a length-control system (Birbaumer & Schmidt, 2010).

In addition to the muscle spindles, the Golgi Tendon Organ (GTO) can be found at the junction between muscle and tendon and lies in series with the extrafusal muscle fibers (Silverthorn, 2010). The series arrangement in association with the very low threshold and high dynamic sensitivity exhibited by the sensory endings provide the CNS with information primarily about active muscle tension (developed during contraction) rather than passive

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tension (developed during inactive muscle stretching, Jami 1992). Therefore, information from the GTO complex is used for tension- control (Birbaumer & Schmidt, 2010).

As with the muscle spindle and tendon organ, joint mechanoreceptors also provide proprioceptive information necessary for sensorimotor control and movement coordination (Silverthorn, 2010). Joint receptors vary in location (e.g. capsule, ligament, and connective tissue), type (e.g. Ruffini, Pacini, free nerve endings) and function. The more slowly adapting Ruffini endings can be categorized as static or dynamic and are capable of detecting joint position and displacement, angular velocity and intra-articular pressure (Johansson, Sjölander, & Sojka, 1991). In contrast, the rapidly adapting Pacinian corpuscles convey the sensation of vibration and mechanical stress as they quickly stop firing to a continuous sensory stimulus (Bell, Bolanowski, & Holmes, 1994). Free nerve endings are widely distributed and feed information into the nociceptive system. Additionally, the cutaneous mechanoreceptors react to pressure (mechanical deformation) and provide information about how we interact with the surroundings (Enoka, 2008).

In neurophysiology the peripheral neural traffic can be quantified by using the method of microneurography. Microneurography is able to record electrical impulses from human peripheral nerves in situ. The first successful recording of neural impulses was conducted by Hensel and Boman who were able to record single unit afferent discharges in healthy humans (Hensel & Boman, 1960). Afferent discharges from muscle spindles, tendon organs, mechanoreceptors and nociceptors can be separately analyzed (Mano, Iwase, & Toma, 2006). Recordings of afferent discharges from muscle spindles are useful to analyze neural mechanisms of sensorimotor control. Especially in rehabilitation, microneurography might be able to confirm afferent information deficits which are assumed to lead to joint instabilities.<sup>6</sup> Recently, an investigation found promising differences in peripheral muscle spindle traffic in unstable ankles compared to healthy controls during ligamentous stress (Needle, Swanik, Farquhar, Thomas, Rose & Kaminski, 2013).

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<sup>6</sup> In a combined EEG and microneurography pilot study we actually examine the changes in afferent muscle spindle traffic and somatosensory cortex activity due to anterior loading of tankles in collaboration with Prof. Swanik (University of Delaware, USA). The project is supported by the DFG (BA 4062/4-1)

Afferent somatosensory inputs are conducted to different areas of the brain by using distinct neural pathways. The major ascending pathways of the spinal cord are the spinothalamic tract, the spinocerebellar tract and the dorsal column (sometimes referred to as the dorsal column-medial lemniscus system). Whereas the spinothalamic tract transmits information about temperature and pain mainly to the thalamus (Gardner & Johnson, 2013b) and the spinocerebellar tract conveys information about limb and joint position and project ipsilaterally to medial zones of the cerebellum (Gardner & Johnson, 2013b), proprioceptive information about touch, vibration, motion, position and tension is transferred via the dorsal column where they are exactly organized according to receptor type, body location and time (Davidoff, 1989) and ends in the somatosensory cortex (Amaral, 2013).

Electrophysiological responses to sensory stimuli can be non-inversely recorded using the electrophysiological method of somatosensory evoked potentials (SEP, Nuwer, 1998). They are used to confirm and localize sensory abnormalities and to monitor changes in neural pathways. Evoked responses are quantified by measuring peak amplitudes and latencies. Nowadays it plays a role in standard clinical examinations (Walsh, Kane, & Butler, 2005), but is also frequently used for research. For example Valeriani et al. (Valeriani et al., 1999) used this method to study knee proprioception and somatosensory evoked potentials (SEPs) after stimulation of the common peroneal nerve at the knee in patients with an ACL injury. They found central nervous system modifications which are located above the medial lemniscus and limited to common peroneal nerve somatosensory pathways. These studies were the initial motivation to look into sensory information processing after ACL injuries and thus one of the foundations of our interest to look into the brain in sensorimotor control.

### **c. The Brain in Sensorimotor Control**

The nervous system is formed by its basic element: the neuron. Neurons are connected via synapses and form neuronal assemblies or networks. These networks are hierarchicly ordered, dynamic in nature and interconnected with other cortical networks. Functions of the

brain originate from specific sets, connections and adaptations of neuronal networks (Kandel, 2013). Sensory information within the complex, sensorimotor system is processed at three distinct levels within the CNS and follows a hierarchy of organization with increasing complexity: the spinal cord, the subcortical regions of the brain and the cerebral cortex (Amaral & Strick, 2013).

At spinal level, sensory information serves two important functions. It is conducted to and from the cerebral cortex and it operates unconsciously with local segmental reflexes supporting movement patterns which are provided from higher levels of the CNS (Pearson & Gordon, 2013).

At the subcortical level, different areas of the brain like the cerebellum, the basal ganglia or the thalamus serve important functions in sensorimotor control. In this way, the cerebellum and the basal ganglia have an essential and distinctive role in coordinating, timing and sequencing of a normal motor output as well as for the motor learning process, but they differ in specific aspects and phases (Groenewegen, 2003). The cerebellum consists of more than one-half of the neurons in the brain and operates between sensory and motor systems, but is not directly involved in the perception of sensation (Enoka, 2008). This region is mainly responsible for the comparison of internal feedback signals (reporting intended movements) with external feedback signals (reporting actual movements). For a repetition of movement the cerebellum is able to provide corrective signals which reduce the error (Lisberger & Thach, 2013). In addition, Rothwell (1994) has described the cerebellum as a timing coordinator and learning device. For a deeper insight Manto et al. (2012) have published an elucidating and helpful consensus statement on the role of the cerebellum in sensorimotor control.

Even if current models are not fully satisfactory it seems to be clear that the basal ganglia, the thalamus and the cerebral cortex act in a family of neuronal networks involved in sensorimotor control. The basal ganglia represent five closely related nuclei. Caudate, putamen, globus pallidus, subthalamic nuclei and substantia nigra have an influence on the motor cortex to shape the final descending signal. The possible role of the basal ganglia in

movement is thought to be in movement initiation and sequencing of movement fragments (Wichmann & DeLong, 2013). The thalamus consists of two egg-shaped masses that lie in the center of the brain. It serves as the main sensory relay for all senses (without smell). The thalamus acts as a gatekeeper for information to the cerebral cortex. It contains connections to nearly all parts of the cortex and the basal ganglia. The ventral lateral posterior nucleus is one of 50 nuclei of the thalamus which seems to be important for sensorimotor control. This part of the thalamus process somatosensory information and project to the primary somatosensory cortex in parietal areas of the brain whereas other portions participate in sensorimotor control by transmitting information from the cerebellum and basal ganglia to the frontal parts of the cortex (Amaral, 2013).

The third and highest level of sensorimotor control occurs in the small layer of cortical neurons in the outer shell of the brain, the cerebral cortex. The cortex is anatomically organized in two hemispheres and represents major sensory and motor areas (Amaral, 2013). Sensory areas are located in the posterior, executive and motor areas of the cortex anterior to the rolandic fissure (Fuster & Bressler, 2012). The German anatomist Korbinian Brodmann defined and numbered the networks of the brain based on the cytoarchitectural organization of its neurons (Figure 2-2; Brodmann, 1925).

The anterior areas of the cortex can be addressed as the “motor” or “executive” cortex in the broadest sense. At the lowest level of the motor hierarchy the primary motor cortex in the precentral gyrus BA4 is responsible for the execution of all voluntary movements. The different areas of the motor cortex represent different body parts, specified as somatotopically organized (Rasmussen & Penfield, 1947). Here, the left hemisphere is specialized for movements of the right body side and vice versa. The area immediately in front of the primary motor cortex is termed the premotor cortex (PMC, BA6). Many studies have drawn attention to the distinct roles the lateral PMC and medial PMC (defined as supplementary motor area, SMA). The PMC mainly acts in more complex movements with objects in the environment (e.g. grasping, reaching), whereas the SMA has been associated with spontaneous well learned action sequences without strong demands in control and

executive functions (Rizzolatti & Kalaska, 2013). At the top of the anterior areas of the cortex is the prefrontal cortex including the dorsolateral prefrontal cortex (DLPFC, BA9/BA46), the anterior prefrontal cortex (BA10) and the orbitofrontal cortex (OFC, BA11/BA47). Based on neuropsychological and brain imaging data the area of the prefrontal cortex provides the broad schemas of action in skeletal and speech domains (Goldman-Rakic, 1995; Fuster, 2000). The prefrontal cortex is also highly interconnected with the thalamus and the anterior cingulate cortex (ACC, BA24) which projects to the posterior parietal cortex. This specific connection will play an important role in the following chapters.

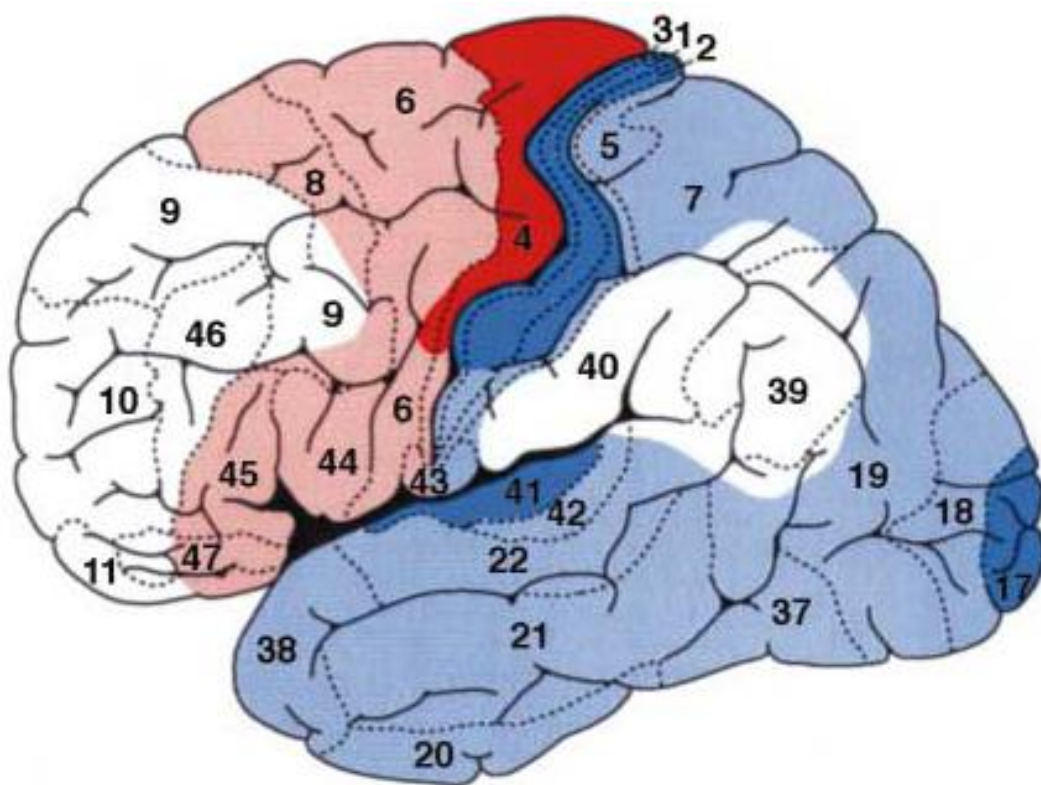


FIGURE 2-2: Brodmann's cytoarchitectonic map of the left hemisphere with the distribution of sensory and motor/executive area anterior (red) and posterior (blue) of the rolandic fissure (RF, Fuster & Bressler, 2012)

The posterior areas of the cortex are responsible for sensory information processing. Even here, Wilder Penfield found in exciting examinations over many years that all parts of the body are represented in the primary somatosensory cortex somatotopically (Penfield &



Boldrey, 1937). The area of the cortex representing each body part is proportional to the density of innervation. Therefore the area devoted to the fingers of the hand is larger compared to the area of the arm. Early stages of processing of somatosensory information takes place in neurons in the primary somatosensory cortex areas BA3a, BA3b and BA1. In sensory processing anterior parietal areas (BA2) are primarily concerned with unimodal sensory modalities. As a principle, unimodal areas (e.g. proprioceptive, auditory, visual) are not interconnected with each other (Yeterian & Pandya, 1985). Multimodal sensory information processing takes place in the posterior association cortex in brain areas BA5 and parts of BA7. Here, representations of the environment are shaped by combinations of different sensory information from somatic, visual and other modalities to guide motor behavior through conceptual representations (Kandel, 2013).

Sensorimotor control with the described components is a key feature for precise goal-directed movements in sports and exercise and the brain obviously plays an important role. However, to our knowledge, there is a dearth of studies which examine brain functions in this context. With new neuroscientific methods it is time to change this and focus should be shifted to the brain in sports medicine research.

#### **d. Looking at the brain in sports and exercise**

In the last decades, brain imaging including Positron Emission Topography (PET) , functional Magnet Resonance Imaging (fMRI) and even electroencephalography (EEG) have developed rapidly and allow new insights into brain functions in excellent spatial (PET, fMRI) and temporal (EEG) resolutions. As a disadvantage nearly all brain imaging experiments are conducted under laboratory conditions. In classic experiments in neuroscience participants had to be quiet and were limited to minimal active motor behavior (like finger movements) while brain activity was obtained. This laboratory condition has nothing in common with the typical sports-related, complex environment. One can argue that effects from the laboratory experiments with minimal movements are not transferable to the ever changing and complex

environment where sports and exercise takes place. Consequently brain measures in a sports related field need to allow for ecologically valid investigations of brain function in real world environments (Makeig, Gramann, Jung, Sejnowski, & Poizner, 2009) and must fit certain requirements like mobility and wearability to be applicable and useful (Gramann et al., 2011) to sports medicine research. Obviously, these requirements lead to methodological approaches like EEG and near-infrared spectroscopy (NIRS). In contrast to PET and fMRI which are heavy and therefore stationary by nature, EEG and NIRS can be used to image the brain during mobile activities. Researchers have used NIRS for measures of oxygenated hemoglobin in different brain areas during locomotion (e.g. Rooks, Thom, McCully, & Dishman, 2010; Suzuki, Miyai, Ono, & Kubota, 2008; Harada, Miyai, Suzuki, & Kubota, 2009) and cycling (e.g. Pei-Yi Lin, Sang-I Lin, & Chen, 2012; Tsujii, Komatsu, & Sakatani) exercise. The NIRS methodology is based on the premise of neuro-vascular coupling and monitors regional tissue oxygenation reflecting perfusion status (Marin & Moore, 2011). However, the limitations of NIRS are controversial about accuracy/reliability (Hoshi, 2003) and the slow dynamics of hemoglobin concentration which provide a temporal resolution of several seconds (Irani, Platek, Bunce, Ruocco, & Chute, 2007). Even if the resolution in time is slow, NIRS might be used as an adequate measure to evaluate brain activity in real world environments.

In our view, a good possibility to measure event-related and ongoing brain activity in goal-directed movements in sports and exercise seems to be the electroencephalography (EEG). The EEG is a non-invasive tool to directly measure electrophysiological brain activity whereas all other methods use an indirect approach by measuring changes in blood flow reflecting e.g. hemoglobin concentration.

Since 20 years approaches have been made with EEG measures in real-world environments in goal directed movements (e.g. Landers et al., 1991; Crews & Landers, 1993; Loze, Collins, & Holmes, 2001; Doppelmayr, Finkenzeller, & Sauseng, 2008; Muangjaroen & Wongsawat, 2012; Del Percio et al., 2011) and exercise (e.g. Mechau, Mücke, Weiss, & Liesen, 1998; Barthel et al., 2001; Brümmer, Schneider, Abel, Vogt, & Strüder, 2011; Hilty, Langer,

Pascual-Marqui, Boutellier, & Lutz, 2011; Gwin & Ferris, 2012; Schneider, Rouffet, Billaut & Strüder, 2013). This field has now advanced with new high-density EEG systems and sophisticated signal processing algorithms (Thompson, Steffert, Ros, Leach, & Gruzelier, 2008; Makeig et al., 2009; Grech et al., 2008; Gramann et al., 2011).

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## CHAPTER 3

### Measuring Brain Activity – EEG as a Valuable Approach

The decomposition of brain activity in sports and exercise calls for a mobile approach for use in a real world environment. Traditional brain imaging techniques like fMRI or PET provide an excellent spatial resolution, but due to their weight these machines/equipment are not portable and can therefore not serve as measuring tools in a sports environment. The EEG is a mobile and light device which can help to analyze cerebral cortical activity over time (Gramann et al., 2011). The signals are sum potentials of excitatory and inhibitory postsynaptic activities, which can be divided in evoked and spontaneous activity. Evoked activity (ERP) correlate change in signal strength and amplitude related to certain events, whereas the spontaneous (or ongoing) activity has been shown to be superior at detecting effects in physiological brain states (Ward, MacLean, Kirschner, & Valdes-Sosa, 2010). Thus, EEG seems to be the best choice for detecting brain states and activity patterns in real world environments related to sports and exercise.

#### a. EEG recording and frequency analysis - A short overview

Billions of neurons form the human brain. Neurons are interconnected and electrical signals known as action potentials play an important role in the communication between neurons. The electroencephalogram is a record of the oscillations of a sum of brain electric potential and reflects the flow of currents through the extracellular space (Nunez & Srinivasan, 2006). These wavelike signals originate not only in the cortex but also in deeper (subcortical) areas in the brain (Nunez & Srinivasan, 2006). Synchronized activity of large numbers of neurons and neural assemblies results in electromagnetic fields that can be measured on the surface of the scalp with sensors (electrodes) embedded in an EEG cap.

The synchronized firing of large neuronal assemblies is believed to be driven by a pacemaker most probably situated in the thalamus (Bruno & Sakmann, 2006) and can be modulated by different cognitive or motor related events, tasks, states and behaviours such as attention, movement and cognition.

Neural synchronization is also thought to play a role in neuronal communication (Fries, 2005; Salinas & Sejnowski, 2001). A change in neural synchronization can be observed by measuring the increases and decreases in activity in the different frequencies of the oscillations produced by the neurons. Increased amplitude shows greater synchronized neurons firing at any one time. Lower amplitude implies that neuronal firing is desynchronized (Figure 3-1; Kirschstein, 2008).

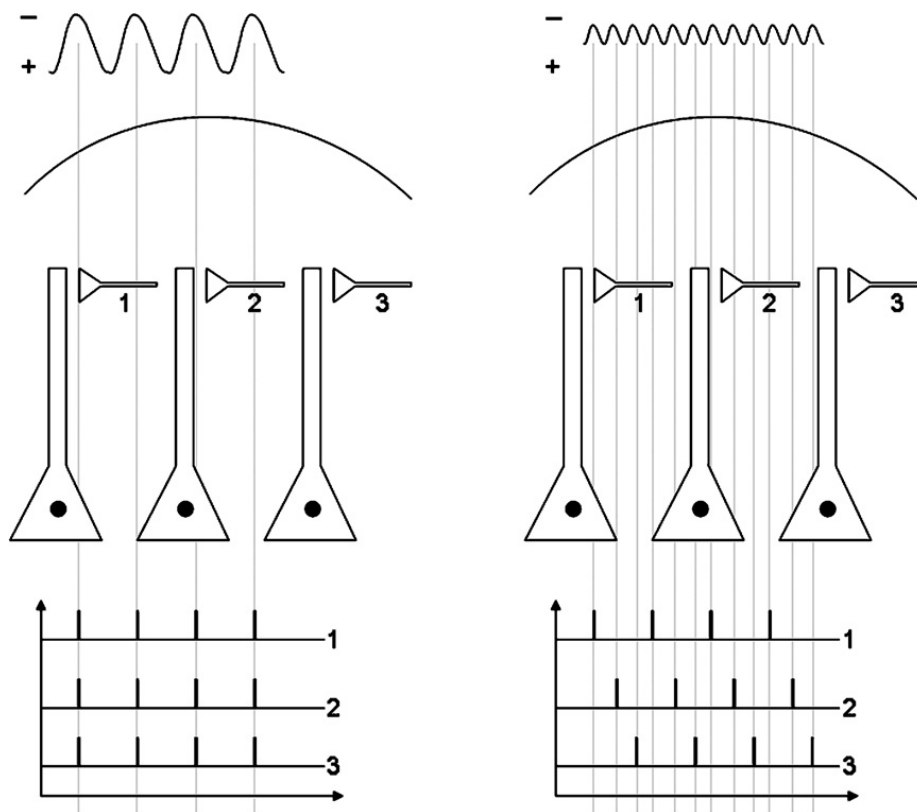


FIGURE 3-1: Model of synchronization and desynchronization in three cortical neurons

EEG measurements can be conducted with a low (from 4) to a high (256) electrode density and the electrode placement is standardized to the international 10-20 system (Klem, Lüders,

Jasper, & Elger, 1999; Figure 3-2) related to a reference montage (e.g. bipolar, common average, Laplacian; Nunez & Srinivasan, 2006). The signals are sampled at a high rate (512 Hz) and amplified due to the microscopic small potential magnitude. The preprocessed signal is carefully filtered to eliminate noise and transmitted to a computer system where it can be processed and spectral parameters can be calculated (Thompson et al., 2008).

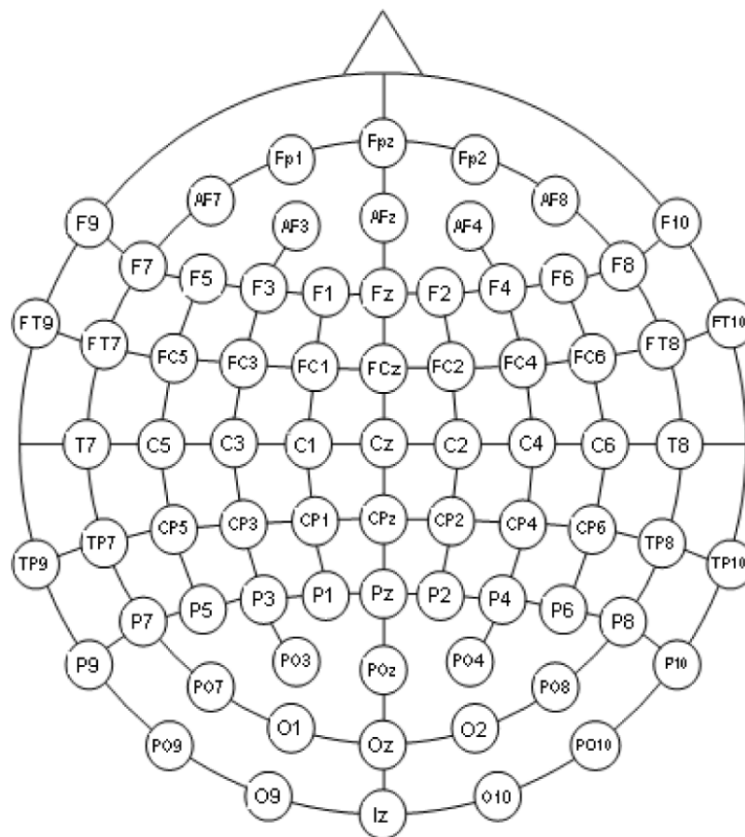


FIGURE 3-2: 10-20 system of electrode placement according to Jasper (1956)

Neuroanatomical distinct areas of the cortex create a variety of different rhythms. To obtain the frequency content of the EEG activity the frequency power spectrum is calculated by applying mathematical calculations and assumptions called a Fast Fourier Transform (FFT). The EEG data are converted from a wavelike display of voltage changes over time to a display of grand average power spectrum. In the frequency power spectrum there is always a trade off between the time- and the frequency resolution. The larger the data epoch length the better the frequency resolution will be and vice versa (Pivik et al., 1993).

The overall EEG power spectrum is decomposed in different frequency bands in the range from 1-45 Hz which have been historically divided into frequency bands defined as Delta (0-3.5 Hz), Theta (4.5-7 Hz), Alpha-1 (7-9.5 Hz), Alpha-2 (10-12.5 Hz), Beta-1 (13-19 Hz), Beta-2 (20-32 Hz) and Gamma (> 35Hz) (Niedermeyer, Schomer, & Lopes da Silva, 2011). The amount of activity is specified by spectral power ( $\mu\text{V}^2$ ) within each frequency band. Thus, EEG power in each frequency at each electrode location is calculated. Depending on the frequency the signal can be distributed widely over the entire scalp area (e.g. Alpha power, Bonnet & Arand, 2001; Niedermeyer et al., 2011) or be related to a distinct location/area (e.g. fronto-midline Theta, Doppelmayr et al., 2008; mu rhythm in sensorimotor areas, Pfurtscheller, Neuper, Andrew, & Edlinger, 1997).

### **b. Reliability of EEG measures**

The EEG measures very small electrical potentials [ $\mu\text{V}$ ] while participants perform cognitive and/or sensorimotor tasks. EEG power values thought to be sensitive to changes in an individual's brain state associated to task changes (e.g. change in information processing, Ward et al., 2010). For example, the posterior Alpha power demonstrates a decrease when eyes are open compared to an eyes closed situation ("Berger Effect"; Berger, 1929). The effect is due to the increase of visual information which leads to a desynchronization of Alpha power. These and other findings in EEG power changes lead to the suggestion that task-related EEG measures can be used to assess temporary, functional changes in brain states due to conditions which are thought to cause acute or chronic changes in the cerebral cortex (e.g. caused by stress-related, sports-related, injury related conditions). A first step in developing EEG-based methods to measure changes in brain states in real world environments associated with sports and exercise is to demonstrate high test-retest reliability in such measures. Reliability is a property which is used to describe the overall consistency of an EEG measure by producing similar results under consistent conditions.

TABLE 1-1: Reliability of EEG power in resting, cognitive and sensorimotor conditions

Study	Population	Task	Test-retest interval	Type of reliability	Classification of reliability
Maltez (2004)	57 healthy adults M: n=23; 23 yrs F: n=34; 22 yrs	Rest (eyes open)	Within-session 15 min (halfsplit)	Pearson-Correlation-Coefficient Theta: 0.93 – 0.98 Alpha: 0.93 – 0.98	high
Cannon (2012)	19 healthy students M: n=12; 20.7 yrs F: n=7; 20.7 yrs	Rest (eyes open/closed)	Between-session 30 days	Intra-class-correlation coefficient Eyes open (topogr.) 0.62 – 0.87 Eyes closed (topogr.) 0.75 – 0.96	high
Gudmundsson (2007)	15 healthy elderly M: n=2; > 50 yrs F: n=13; > 50 yrs	Rest (eyes closed)	Between-session 60 days	Intra-class-correlation coefficient Theta: 0.87 – 0.91 Alpha-1 0.67 – 0.85 Alpha-2 0.81 – 0.88	high
Gasser (1985)	26 healthy children M: n=10; 10-13 yrs F: n=16; 10-13 yrs	Rest	Between-session 300 days	Rank-correlations Theta 0.57 – 0.79 Alpha-1 0.74 – 0.89 Alpha-2 0.64 – 0.84	moderate to high
McEvoy (2000)	20 healthy adults M: n=9; 18-29 yrs F: n=11; 18-29 yrs	Rest	Within-session	Pearson-Correlation-Coefficient Theta 0.74 – 0.87 Alpha-1 0.92 – 0.93 Alpha-2 0.82 – 0.93	high
		Rest	Between-session 7 days	Pearson-Correlation-Coefficient Theta 0.76 – 0.86 Alpha-1 0.9 – 0.95 Alpha-2 0.77 – 0.89	high
		cognitive task (WMM)	Within-session	Pearson-Correlation-Coefficient Theta 0.92 – 0.97 Alpha-1 0.96 – 0.99 Alpha-2 0.93 – 0.98	high
		cognitive task (WMM)	Between session 7 days	Pearson-Correlation-Coefficient Theta 0.83 – 0.93 Alpha-1 0.9 – 0.97 Alpha-2 0.93 – 0.97	high
Baumeister (2012)	20 healthy adults M: n=20; 24.2 yrs	motor task (JPS)	Between-session 7 days	Intra-class-correlation coefficient Theta: 0.87 – 0.95 Alpha-1 0.89 – 0.97 Alpha-2 0.90 – 0.97	high



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Interestingly there is only limited published data on EEG test-retest studies. Studies have demonstrated reliable results in resting (Gasser, Bächer, & Steinberg, 1985; Maltez, Hyllienmark, Nikulin, & Brismar, 2004; Gudmundsson, Runarsson, Sigurdsson, Eiriksdottir, & Johnsen, 2007; Cannon et al., 2012), cognitive (McEvoy, Smith, & Gevins, 2000) and sensorimotor (Baumeister et al., 2012) tasks in different periods over the lifespan (Table 1-1).

Most of these studies demonstrated a high test-retest reliability within- and between-sessions. Only Gasser et al. (Gasser et al., 1985) show a moderate to high reliability in Theta power (rank-correlation 0.57-0.79) which might be due to the specific subgroup (children) they used. Children are in a developing period especially of frontal brain functions where Theta is involved and demonstrates a higher variability in EEG power (Michels et al., 2012). Furthermore this study chooses a frequency range from 3-7 Hz for calculating Theta power. The lower level might overlap the delta frequency which is known to be much more variable compared to other frequencies.

The studies where the participants have to perform a cognitive or sensorimotor task demonstrate a higher reliability compared to the resting conditions in other studies (McEvoy et al., 2000; Baumeister et al., 2012). McEvoy et al. (McEvoy et al., 2000) explained this result with higher variations in attention and alertness in resting states.

This short review of the EEG reliability data leads to the conclusion that, under certain conditions, EEG power measures in a resting state or associated with the performance in a cognitive or sensorimotor task, are highly reliable. These findings suggest that sensorimotor tasks have the necessary reliability for use in assessing transient changes in the neurophysiological signals of sensorimotor control due to different conditions and subpopulations.

### c. EEG in laboratory and real world environments

A considerable problem in EEG measurements is to get clean data from cerebral sources which are not contaminated by artifacts (Thompson et al., 2008). Sports and exercise occur in a complex real-world environment and different conditions like e.g. motion and sweating. These surroundings often cause physiological (muscular and eye blink artifacts) and physical artifacts (e.g. cable sway, electrode movement)<sup>7</sup>. At first glance, this does not seem to fit the requirements to get valid, microscopic small and sensible EEG signals [in  $\mu\text{V}$ ].

Two complementary approaches are possible to reduce or eliminate artefacts in sports-related environments. The first can be described as “minimizing head movements” during the recording stage (Thompson et al., 2008). Therefore, most of the published experiments which include EEG measures use situations during a performance where head movements are controllable, e.g. golf (Crews & Landers, 1993; Babiloni et al., 2011; Zhu, Poolton, Wilson, Maxwell, & Masters, 2011; Mann, Coombes, Mousseau, & Janelle, 2011), shooting (Haufler, Spalding, Santa Maria, & Hatfield, 2000; Haufler et al., 2000; Loze et al., 2001; Doppelmayr et al., 2008; Hung, Haufler, Lo, Mayer-Kress, & Hatfield, 2008; Liu, Wang, Jin, & Song, 2010; Paes et al., 2011), stationary bike cycling (Fumoto et al., 2010; Hilty et al., 2011; Brümmer et al., 2011; Schneider, Rouffet, Billaut, & Strüder, 2013) or in a pre-post design before (preparation) and after (recovery) exercise (Mechau et al., 1998; Barthel et al., 2001; Nielsen & Nybo, 2003; Woo, Kim, Kim, Petruzzello, & Hatfield, 2010) where the head movements can be diminished.

The second approach requires processing of the obtained EEG data to separate neural signals from artifacts through computational methods. Post processing algorithms depend on the signal dimensions (Thompson et al., 2008). The number of channels is a dimension which impacts the relative strength of EEG quantification. More channels carry more information which affects the robustness and therefore the reliability of the data significantly. On the other hand a high dimension produces a high amount of data which limits the artifact

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<sup>7</sup> For a detailed review see Nunez & Srinivasan 2006

rejection procedures to offline processing. Even if all electrode positions carry information sometimes a distinct region of interest or frequency band reduce the chance of artifacts. For example, the sensorimotor area is less affected by cranial muscle activity as well as by a major contamination source, the eye blinks. Also frequency bands can be isolated to ranges which are more robust to artifacts, such as Theta and Alpha frequencies in the range of 4-13 Hz. This range is less affected by typical low frequency (e.g. movements, eye blinks, sweat) and high frequency (e.g. muscle activity, electrical noise) artifacts. Hence, the choice of cortical areas and EEG frequencies can contribute to the quality of EEG measures of sensorimotor control in sports and exercise.

All these procedures cannot hide the fact, that EEG measures are prone to artifacts which demand post processing of the signals. The usual approach over the years included automated as well as manual procedures. The offline pre-processing includes automatic rejection of signals according to eye-blinks (Semlitsch, Anderer, Schuster, & Presslich, 1986) and the slope and magnitude of the signals. Although some artifacts can be identified by automated rejection mechanisms, these procedures will miss a substantial portion of contaminating artifacts. Therefore, careful manual screening by highly trained and experienced electroencephalographers is mandatory in EEG signal processing. Modern processing involves spatial filter algorithms like independent component analysis (ICA) to solve the problem of volume conduction and to distinguish between brain and non-brain related cortical activity (Delorme & Makeig, 2004; Delorme et al., 2011).

Given all these possibilities of recording and processing electrophysiological signals, EEG spectral data is a valuable approach to brain states in a real world environment. Therefore the EEG frequency analysis is a possible method of choice to elucidate brain mechanisms in sensorimotor control in sports and exercise.

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## CHAPTER 4

### **Between perception and action – the working-memory-hypothesis in sensorimotor control**

After the description of the physiological components which are involved in sensorimotor control (Chapter 2) it is clear that the brain and especially the cortex play an important role in organizing sensorimotor control. The human cortex is formed by millions of neurons which are connected to networks. Neurons communicate within and between neuronal assemblies or networks using electrophysiological signals (Koester & Siegelbaum, 2013; see also Chapter 2 and Chapter 3).

The term “sensorimotor” implies that two broad, widely distributed networks exist which are responsible for sensory and motor processing. In the literature these terms are often specified as perception and action networks. Perception is referred to as ‘perception circuits’ (sensory part) posterior and action networks are referred to as ‘action circuits’ (executive/motor part) anterior to the central sulcus (Fuster & Bressler, 2012). The basic principle of distributed perception and action circuits in posterior and anterior parts of the brain in its originally neurobiological form involves feedback from the environment processed for goal-directed motor behavior and goes back to Jakob Johann Uexküll, a German biologist who described this biokybernetic cycle to higher animal species (Uexküll, 1926). Later, it had also been applied to neurology (Weizsäcker, 1950) and computational neuroscience (Aribib, 1981).

The circuits are hierarchically organized. Perception circuits range from unimodal sensory processing to sensory representations in parietal association areas (Kandel, Barres, & Hudspeth, 2013) whereas the action circuits include motor and executive processes and representations in prefrontal association areas (Fuster, 2006). Conceptual representations are memories and knowledge based on individual experience. It is widely accepted that

representations are organized by the modulation of synaptic transmission according to Hebbian principles (Fuster, 2006; Kandel et al., 2013).

An important brain function which interconnects both circuits is the working memory (Fig. 4-1). The Working memory temporarily maintains and stores information and acts as an interface between the perception and action circuits (Baddeley, 2012). The british psychologist Baddeley placed the working memory in a fronto-parietal network with sensory subsystems (phonological loop, visuo-spatial sketchpad) located in the parietal perception circuits and the executive control in prefrontal action circuits (Baddeley, 2003). It is suggested that executive action allocate attentional resources to the subsystems in perception circuits (Schacter & Wagner, 2013).

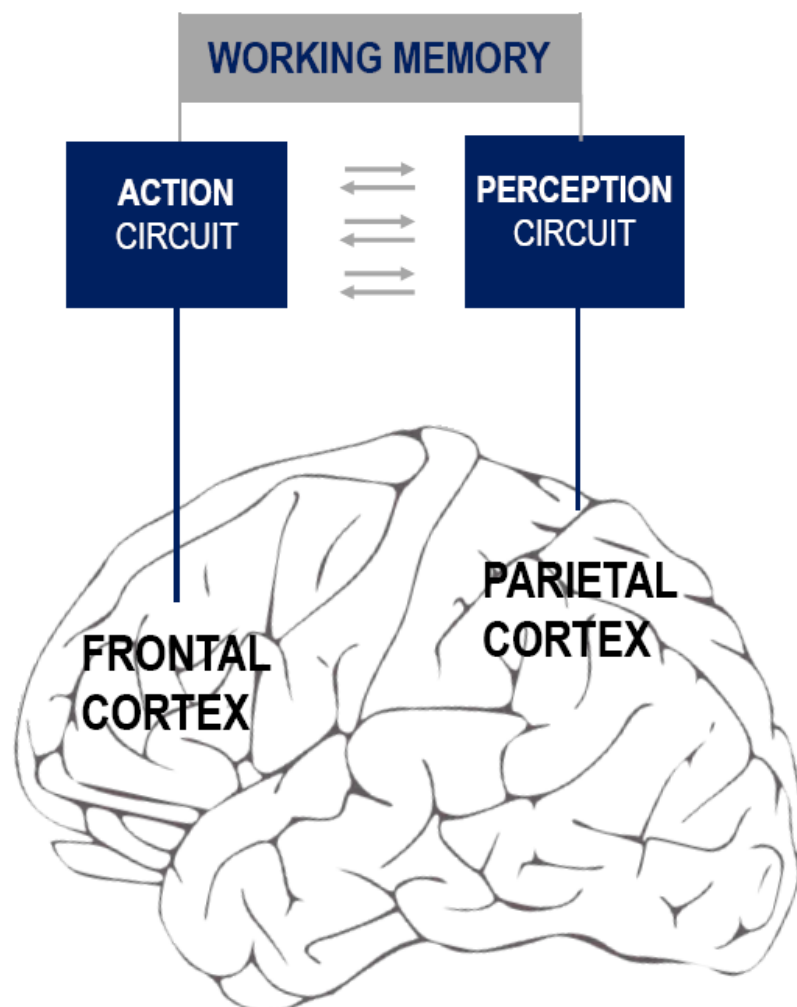


FIGURE 4-1: Working Memory as an interface between perception and action

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Activity in the perception circuit is specified as a sum of electrophysiological signals of many processes in the parietal association area which can be measured by EEG as a “final common activity”, but cannot distinguish between distinct processes. The same can be applied for action circuits in prefrontal association areas.

A high amount of research has demonstrated that EEG Alpha-2 power is a possible marker for processing brain activity in the perception circuit whereas EEG Theta power is suggested as a possible marker for processing brain activity in action circuits closely related to an attentional control network. The following section will provide evidence for these relationships.

The Theta frequency is an EEG signal in the range from around 4-7 Hz described as prominent in frontal regions of the cortex. In the last decade human frontal Theta activity had been consistently described to play a major role in working memory functions including memory processes, information processing and attention in cognitive tasks. There is electrophysiological evidence from combined EEG-fMRI studies that this signal is most likely generated in the anterior cingulate cortex (ACC; Gevins, Smith, McEvoy, & Yu, 1997; Schacter & Wagner, 2013). The ACC is thought to be part of the human attentional control network involved in target selection, error detection and performance monitoring (Posner & Rothbart, 2007). This is consistent with the results from other neuroimaging studies (LaBerge & Buchsbaum, 1990; Vogt, Finch, & Olson, 1992), a monkey model (Tsujimoto, Shimazu, & Isomura, 2006) and brain lesion studies (Janer & Pardo, 1990) suggesting the ACC as a part of the human attentional control system. In an expanded explanation Sauseng et al. (2005) described the dynamics of the frontal Theta frequency as a general brain integrative mechanism which is relatively specific for control of working memory processes. Thus, the frontal Theta power is assumed to be sensitive for attentional control with an increase in Theta power related to higher attentional demand.

It is also widely accepted that the Alpha-2 component tends to be sensitive to processing demands in parieto-occipital regions in the area of the somatosensory cortex (Gevins et al., 1997; Smith, McEvoy, & Gevins, 1999; Neuper, Wörtz, & Pfurtscheller, 2006). A suppression

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of Alpha-2 power, interpreted as 'desynchronization', suggests that a large population of neurons in this specific area no longer oscillate in synchrony. This desynchronization (a decrease of Alpha-2 power) in parietal brain areas is known according to information processing demands due to incoming sensory information (Klimesch, 1997). Vice versa a 'synchronisation' is described related to a deactivation / inhibition of neuronal populations in parietal brain areas (Neuper et al., 2006). To conclude, parietal Alpha-2 power is assumed to be sensitive to variations in sensory information processing demands.

Since twenty years the concept of the working memory as an interface between perception and action (Baddeley, 2003) is under investigation using the ongoing EEG. Numerous studies have reported that oscillatory power in the Alpha band (8–12 Hz) over posterior sites in perception circuits (Jensen, Gelfand, Kounios, & Lisman, 2002; Schack & Klimesch, 2002; Tuladhar et al., 2007; Scheeringa et al., 2009) and in the Theta band (4–8Hz) in prefrontal action circuits (Gevins et al., 1997; Jensen & Tesche, 2002; Meltzer, Negishi, Mayes, & Constable, 2007; Meltzer et al., 2008) are correlated with working memory load and attentional demands in cognitive tasks which rely mainly on visual sensory information.

The evidence about working information processing and attentional demands in sensorimotor control concerning afferent proprioceptive information is considerably smaller, but also demonstrates similar variations in EEG power in perception and action circuits (Smith et al., 1999; Grunwald et al., 2001; Slobounov, Fukada, Simon, Rearick, & Ray, 2000). All of these visio-motor tasks involve only hand coordination in sitting position in a laboratory setting. Instead, research in sensorimotor control and associated brain activity in the field of sports and exercise should involve typical conditions and real world environments. Thus, a research program is needed which satisfied this needs.

*However, relevant data and supportive evidence from neuroscience are able to establish a theoretical basis for the working-memory-hypothesis in sensorimotor control. The working-memory-hypothesis proposes that EEG power in perception and action circuits is sensitive to changes in neural processing demands due to afferent sensory information and attentional control.*

## CHAPTER 5

### Research Program and Objectives

The knowledge of brain activity might allow insights into the working-memory-hypothesis in sensorimotor control in the field of sports & exercise and is able to provide a theoretical basis which is testable by scientific standards.

A huge amount of papers in the last two decades demonstrate the sensitivity of EEG power in parietal and frontal brain areas associated with variations in afferent information and attentional demands in cognitive control tasks (Gevins et al., 1997; Jensen et al., 2002; Jensen & Tesche, 2002; Schack & Klimesch, 2002; Tuladhar et al., 2007; Meltzer et al., 2007; Meltzer et al., 2008; Scheeringa et al., 2009). However, only limited knowledge is available concerning the electrocortical brain activity affecting the working memory in sensorimotor tasks (Smith et al., 1999; Grunwald et al., 2001; Slobounov et al., 2000) and there is no evidence which demonstrates the sensitivity of EEG power in more complex conditions and real world environments in the field of sports and exercise.

Therefore, the overall purpose/aim of this research program is:

- (1)** to demonstrate the feasibility of EEG measures associated with sensorimotor control in the environment of sports & exercise and
- (2)** to provide evidence for the sensitivity of EEG power in parietal perception and frontal action circuits related to the working-memory-hypothesis.

Different typical conditions from sports and exercise will help to elucidate the EEG power changes concerning the working-memory-hypothesis which are stress related, sports-related and injury related conditions.



### **a. Sensorimotor control and brain activity in stress-related conditions**

Stress-related conditions are a typical object of investigation in sports and exercise. When physical or mental stresses induce a decline in performance it is classified as fatigue. Fatigue is very complex and cannot be explained by a single mechanism. It may arise not only because of peripheral changes at the level of the muscle, but also because the central nervous system (CNS) fails to drive the motor neurons adequately. As many sites within the muscle controls force, so many sites within the CNS can modify the output of motor neurons (Gandevia, 2001).

Many studies have reported that precision in sensorimotor control which is driven by proprioceptive modalities can be disturbed by induced stress. A significant decrease in the subject's ability to reproduce joint angles (e.g. knee, elbow) actively after a sports related fatigue protocol (Skinner, Wyatt, Hodgdon, Conard, & Barrack, 1986; Lattanzio & Petrella, 1998) and due to muscle fatigue (Givoni, Pham, Allen, & Proske, 2007; Walsh, Allen, Gandevia, & Proske, 2006) had been found. But there seems to be a lack of knowledge about feasible mental fatigue protocols and their influence on accuracy in sensorimotor control.

Although the effects of stress-related declines on motor behaviour are described in detail, there seems to be a dearth of investigations which look into the neural mechanisms behind these behavioural changes. The proposed working-memory-hypothesis of this research program examines changes in brain activity related to physical and mental fatigue. In an extension of this idea the working-memory-hypothesis should also be investigated in complex sports-related sensorimotor tasks which are extensively used in all kinds of sports and exercise. Landing from a jump is such an example. From a sports medicine perspective it is a motor action which is prone to injuries in fatigued conditions (McLean et al., 2007; Chappell et al., 2005). But precise injury mechanisms remain unclear and no investigations have ever looked into the neural mechanisms related to landings even if it can be assumed that they play an important role.

Thus, the aims of the research program concerning stress-related conditions in sports and exercise are:

- (1) to demonstrate changes in motor behavior and EEG measures in perception and action circuits in sensorimotor matching tasks due to **different physical and mental stress protocols**.
- (2) to demonstrate changes of EEG measures in perception and action circuits in a sports related sensorimotor task due to **different physical and mental stress protocols**.

From the view of sports medicine the results (Chapter 6) will help to elucidate neural mechanisms of sensorimotor control which are involved in stress-related conditions. As soon as a mechanism can be verified, principles for practice applications in sports and exercise can be derived and will be able to guide training programs.

#### **b. Sensorimotor control and brain activity in sports-related conditions**

Expertise leads to success in sports and exercise. Expertise has been defined as the ability to consistently demonstrate superior performance (Janelle & Hillman, 2003). Superior performance refers to the ability to identify and acquire environmental information (perception) for appropriate responses which are selected and executed (action) (Marteniuk, 1976). Expert sport performers must be superior in sensorimotor control and be able to identify the most relevant information, direct their attention appropriately, and extract meaning from these areas efficiently and effectively (Williams, Davids, & Williams, 1999). Since twenty years researchers are investigating the neural mechanisms responsible for sports expertise. The model of choice had been pistol shooting (Hatfield, Haufler, HUNG, & Spalding, 2004), but marksmanship is a visual-spatial cognitive task with only minimal movement (trigger pull) and therefore is not a good example of a complex sports-related motor task guided by sensorimotor control. The putting performance in golf is a complex, goal-directed motor task which seems to offer the possibility of obtaining reliable EEG data

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during performance. Crews & Landers (Crews & Landers, 1993) conducted the first (and till 2008 the only) study where EEG data was obtained during putting performance in a laboratory setting. It was a rudimentary description of differences in brain activity according to the hemispheres, but this approach was not really productive in elucidating the neural mechanisms behind expertise in golf putting.

Although golf putting in a laboratory setting is able to provide insights into neural mechanisms of sports expertise, nothing is known about these mechanisms in different environments especially in a real-world environment. This research program investigates changes in EEG brain activity based on the proposed working-memory-hypothesis.

Therefore the aims of the research program concerning stress-related conditions in sports and exercise are:

- (1) ... to demonstrate changes in motor behavior and EEG measures in perception and action circuits during a golf putting task due to **different skill levels**
- (2) ... to demonstrate changes in motor behavior and EEG measures in perception and action circuits during a golf putting task due to **different environments**

From a sports medicine perspective the outcome of these studies (Chapter 7) may help to choose a training environment and to develop instruments for the evaluation of the training process which are based on neural measures. In a holistic view, this can add to the standardized cardiovascular and neuromuscular parameters in sport medicine. Thus, training can be optimized in terms of quality and intensity which might also be helpful to avoid overtraining states in sports and exercise.

### **c. Sensorimotor control and brain activity in injury-related conditions**

Most of the research in musculoskeletal rehabilitation is based on a biomechanical approach demonstrating differences between patients and controls in kinetic, kinematic and

neuromuscular data. Motor behavior is described as the final common output of human sensorimotor control after musculoskeletal injuries and damages.

Declines in performance have been demonstrated after ACL injuries in many investigations. A deficient or reconstructed ACL causes biomechanical alterations (Alkjaer, Simonsen, Peter Magnusson, Aagaard, & Dyhre-Poulsen, 2002; Chmielewski, Rudolph, Fitzgerald, Axe, & Snyder-Mackler, 2001), as well as decreases in muscular strength (Elmqvist, Lorentzon, Långström, & Fugl-Meyer, 1988; Baumeister & Weiss, 2002) and function (Rudolph & Snyder-Mackler, 2004; Ageberg, Thomeé, Neeter, Silbernagel, & Roos, 2008). With regards to impaired sensorimotor function after ACL injury/reconstruction studies have also demonstrated deficits in motor behavior using sensorimotor tasks which are closely related to the sensation of joint position, force and motion (Gokeler et al., 2012; Ingersoll, Grindstaff, Pietrosimone, & Hart, 2008). These deficiencies are thought to be related to a disturbance in sensorimotor control.

On a theoretical basis different authors hypothesize that the deficiency in motor behavior is related to a change in the reception of afferent proprioceptive information due to receptor damage in the knee caused by ACL injury or reconstruction which most likely lead to modifications in the CNS. But there is a dearth of studies who claim to investigate these changes in the brain.

Damage in the musculoskeletal system is often accompanied by pain. Pain is another somatosensory sensation which can easily be induced using an exercise induced muscle damage (EIMD) protocol (Brentano & Martins Krueel, 2011). EIMD occurs after unaccustomed exercise where muscles lengthen under tension (Plattner, Baumeister, Lamberts, & Lambert, 2011). Thus the EIMD model provides an interesting model to investigate the effect of pain related to the working-memory-hypothesis in sensorimotor control.

Therefore the aims of the research program concerning injury-related conditions in sports and exercise are:

- (1) to demonstrate changes in motor behavior and EEG measures in perception and action circuits during sensorimotor tasks due to somatosensory modalities (proprioception) after the **damage of the anterior cruciate ligament**
- (2) to demonstrate changes in motor behavior and EEG measures in perception circuits during a sensorimotor task due to somatosensory modalities (pain) after **exercise induced muscle damage**

Injuries are a typical topic in sports medicine research. Cortical alterations in injury-related conditions associated with sensorimotor control (Chapter 8) can help to identify underlying mechanisms. With these results it might be possible to evaluate training/therapy. This can build a scientific base for optimized rehabilitation programs with the understanding why the programs work.

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## CHAPTER 6

### **Sensorimotor Control and Brain Activity in stress-related conditions**

Physical and mental stress is a classical topic in sports and exercise. It is well established that stress influences cognitive and sensorimotor control. But the underlying mechanisms are not well explained.

The results of three publications in this research program demonstrate that stress-related conditions cause variations in EEG power according to the working-memory-hypothesis in sensorimotor feedback and feedforward tasks.

A knee angle reproduction task (feedback) was conducted to quantify precision before, directly after and after 60 minutes of recovery (Publication 1)<sup>8</sup>. Brain activity was obtained during the task and EEG spectral power was calculated. The exhaustive physical stress protocol required the participants to cycle at 70% VO<sub>2</sub>max for 60 min followed by an incremental test. Fatigue was verified by a decrease in maximal voluntary contraction (MVC) peak torque. The exhaustive exercise leads to significant decrease in accuracy in the reproduction task and is accompanied by EEG power changes assumed in the working-memory-hypothesis. The most prominent finding demonstrates a significant decrease in frontal Theta power and also a significant decrease of parietal Alpha-2 power. Motor behavior and brain activity recover 60 min after the end of the exhaustive exercise protocol. The results demonstrate (probably for the first time!) directly measured evidence for altered central brain functions following prolonged exhaustive exercise.

A functional fatigue protocol was induced before and after drop landings which serve as a complex sports-related task (Publication 2). The participants were asked to concentrate on the landing preparation for 10 seconds in a series of drop landings, before an auditory signal

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<sup>8</sup> The study design was also used with a 60 minutes muscle fatigue protocol (PhD thesis, Reinecke 2013) and a 60 min mental fatigue protocol. The data were analyzed and the manuscript is in preparation for The *Scandinavian Journal of Medicine and Science in Sports and Exercise* and *Neuroscience Letters*

requires them to drop land from a 30 cm platform. The induced standardized fatigue protocol consists of 8 sets of 15 vertical jumps with 30 seconds rest between each set. The participants were deemed to be fatigued when they were not able to reach 80% of their maximal jumping height anymore. While the landing biomechanics during impact remained unchanged the functional fatigue protocol leads to EEG power changes related to the working-memory-hypothesis. Frontal Theta power was increased during preparation compared to rest, but do not change after the fatigue protocol. Parietal Alpha-2 power demonstrated higher values in preparation and a further increase of power values in the fatigue condition. Changes in parietal areas may be related to an inhibition of incoming afferent information in the somatosensory cortex which might be specific to a feedforward task. Such a task works with offline (memorized) proprioceptive information rather than incoming peripheral information (inhibition demonstrated by an increase in EEG power). In further studies it will be important to have a deeper look into these feedforward mechanisms compared to online feedback tasks.

To our knowledge there was no evaluated mental fatigue protocol available. Therefore, there is a need for pilot data about performance and electrocortical brain activity of mentally induced stress to develop a standardized mental stress protocol. In a series of cognitive tasks which lasted 30 minutes EEG was obtained in 16 subjects ([Publication 3](#)<sup>9</sup>). The task series consists of a stress inducing delayed auditory feedback task, a stroop color-word task and a d2 task. Frontal Theta power increased from the beginning to the end of the test series indicating higher attentional demands according to the working-memory-hypothesis, but changes were only evident over the prolonged test series and not before and after each single test. Therefore prolonged mental stress is needed to affect attentional control in a mental stress protocol.

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<sup>9</sup> After different pilot studies we now use the continuous performance task (CPT) for 60 minutes which we evaluated for a decrease in performance and associated brain activity in perception and action circuits in a masters thesis (Koutsandreou 2009). This data is in preparation for publication in *Physiology & Behavior*

All stress related conditions affect the brain state according to the working-memory-hypothesis in terms of sensitivity of EEG power after different fatigue protocols. EEG power analysis is also able to show brain activity pattern related to the working-memory-hypothesis in a complex sports related task. There are studies needed which systematically investigate the effect of local, global and mental fatigue protocols on sensorimotor control. Especially in the field of mental fatigue there is a dearth of studies which examines the effects on sensorimotor control. As soon as we have an overview about the effects regarding global, local and mental fatigue on sensorimotor control and associated brain activity we may be able to verify the working-memory-hypothesis and start to propose a model which may have an important impact on injury risk management, prevention strategies and optimizing performance.



## PUBLICATION 1

Baumeister, J., Reinecke, K., Schubert, M., Schade, J., & Weiss, M. (2012). Effects of induced fatigue on brain activity during sensorimotor control. *European Journal of Applied Physiology* 112(7), 2475-82

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Author Contributions:

<b><i>Study concept and design:</i></b>	<b>Baumeister, Weiss</b>
<b><i>Acquisition of data:</i></b>	<b>Baumeister, Reinecke, Schubert, Schade</b>
<b><i>Analysis and interpretation</i></b>	<b>Baumeister, Reinecke</b>
<b><i>Drafting the manuscript</i></b>	<b>Baumeister</b>
<b><i>Critical intellectual revision</i></b>	<b>Reinecke, Schubert, Schade, Weiss</b>
<b><i>Study supervision</i></b>	<b>Baumeister, Weiss</b>

## ABSTRACT

**PURPOSE:** The aim of this study was to look if accuracy in sensorimotor control and cortical activity were influenced after induced fatigue during a knee joint reproduction task.

**METHODS:** 12 volunteers performed a sensorimotor task before and directly after and sixty minutes after a prolonged exhaustive exercise protocol. The task consisted of an active reproduction of a target knee angle. After three practice trials visual feedback was taken away and the task was performed for 10 repetitions at own pace. Reproduction accuracy was analyzed and EEG raw data was obtained from frontal, central, temporal, parietal and occipital scalp locations during the task. Average power spectra in Theta and Alpha frequencies were computed across conditions in each participant.

**RESULTS:** Task accuracy decreases significantly related to fatigue and increased after recovery. This is accompanied by a significant decrease in frontal Theta, Alpha-1 and Alpha-2 frequencies after inducing fatigue. The power values in all frequency bands recovered after 60 min.

**CONCLUSIONS:** Sensorimotor control was influenced by induced fatigue which could be demonstrated in behavior and brain activity. Characteristics in brain activity demonstrated an increase in Theta and a decrease in Alpha-1 and Alpha-2 frequency band power. The changes were discussed related to attentional recourses, alertness and somatosensory information processing mechanisms.

**Key words:** exhaustive exercise, proprioception, joint position sense, EEG, cortical activity

## INTRODUCTION

Control of human movement is based on sensorimotor control, a dynamic interaction between sensations of sensory stimuli, the processing of information in the central nervous system (CNS) and motor behavior. Proprioception plays an important role in detecting and integrating information about motion, acceleration, force and position within the sensorimotor system and is defined as the cumulative neural input to the CNS where muscle spindles, mechanoreceptors such as Ruffini endings, Pacini corpuscles and Golgi tendon organs are responsible for sensations (Sjolander et al. 2002). Afferent proprioceptive information is integrated in motor planning and programming in the CNS and provides feedback for sensorimotor control of goal-directed movements (Riemann and Lephart 2002).

It is generally accepted that fatigue affects sensorimotor control and therefore acuity in motor behavior. The loss of precision leads to a decrease in performance and an increasing risk of injury which is fundamental to sports and exercise. But how does fatigue exactly affect sensorimotor control?

Several authors report a deficit during matching tasks in knee proprioception due to fatigue and have demonstrated a significant decrease in precision in young healthy (Givoni et al. 2007; Van Tiggelen et al. 2008) and elderly (Ribeiro et al. 2007) subjects.

The findings of Lattanzio et al. (1997) who used an exhaustive exercise protocol to induce fatigue demonstrate a significant increase in knee position matching error. The authors propose that fatigue may disturb the acuity of movement due to the impact of metabolites and inflammatory substances on discharge rates of muscle spindles which change the afferent information upstream flow which needs to be processed in the brain. This plausible mechanism indicates that a combination effect of peripheral and central components may be responsible for the perturbation of sensorimotor control.

Whereas it is clear, that fatigue conditions affect the precision of motor behavior there is only limited knowledge about the underlying central mechanisms. It has been demonstrated that a central electrophysiological approach using the Electroencephalography (EEG) is able to

detect changes in brain activity. Numerous experiments have established the sensitivity of EEG measures to different conditions during sensorimotor tasks. Slobounov et al. (2001) investigated the effect of time pressure on a visuomotor tracking task. The results clearly show that time pressure lead to an increased error in motor behaviour and increased activation levels in Theta and Alpha frequency bands. Fournier et al (1999) found an increased error in a visuomotor task with an increasing level of difficulty which was accompanied by a higher activation level in the brain also demonstrated in Theta and Alpha frequency bands. Results from our laboratory (Baumeister et al. 2008a) have demonstrated comparable results in a visuomotor knee angle reproduction task. We have shown that cortical alterations in patients after a peripheral injury (ACL) compared to healthy controls were accompanied by prominent changes in the Theta- and Alpha frequency band.

All these changes in behavior and brain activation demonstrate that an increase of task demand due to a more complex condition (e.g. changes induced by time pressure, difficulty, and injury) is followed by prominent changes in Theta and Alpha frequencies suggesting that the brain has to increase activity workload to solve the given task.

Fatigue may also increase workload activity during the sensorimotor task due to the fact that afferent information is changed and it is assumed that this might increase complexity and result in a decreased acuity in motor behavior and an increased activation in Theta and Alpha frequencies.

The purpose of this pilot study is to characterize the brain states measured in a healthy young population during a knee position matching task involving a prolonged exhaustive exercise protocol to induce fatigue. It was hypothesized that (1) sensorimotor function demonstrate a decrease in performance (matching precision) and (2) the electro-cortical activity (spectral power) demonstrate changes in Theta and Alpha frequency bands related to different fatigue conditions before, directly after and after sixty minutes of recovery.

## METHODS

**Participants.** Twelve healthy male subjects (TABLE 1) volunteered to participate in the study and completed all study requirements. Medical history, a questionnaire, a physical examination and a resting ECG were conducted and evaluated by an experienced medical doctor before the first exercise exposure to ensure an unimpaired health status. Therefore it had been verified that the participants had no history of neurological, cardiovascular or other major disorders and no current use of medications or drugs. They had been asked to have no physical or psychological exposures (e.g. intensive training sessions, examinations) 24 hours prior to the exercise trials. All participants had normal or corrected to normal vision. Written informed consent was obtained from each participant, and the experimental procedures were approved by the ethics committee of the medical association of Westphalia and the Faculty of Medicine at the WWU Munster (AZ 2009-041-f-S).

TABLE 1. Anthropometric and ergometric data

	Age [years]	Height [m]	Weight [kg]	BMI [kg · m <sup>-2</sup> ]	VO <sub>2peak</sub> [mL · min <sup>-1</sup> · kg <sup>-1</sup> ]
<b>Mean</b>	24.0	1.84	83.0	24.4	43.3
<b>(SD)</b>	(2.3)	(0.08)	(8.1)	(1.9)	(5.8)

BMI (body mass index), VO<sub>2peak</sub> (peak oxygen uptake), N=12

**Preliminary testing.** In a preliminary examination the participants were familiarized with surroundings, the task and the procedures one week before the experiment. After that peak pulmonary oxygen uptake (VO<sub>2peak</sub>) was determined during an incremental cycling test. The test protocol started with cycling at 50W and the workload was increased by 50 W every 3 min until exhaustion. The subjects were instructed to pedal at a constant cadence between 70-90rpm. Capillary whole-blood samples for lactate measurements (enzymatic-ampometric method, Eppendorf, Germany) were taken at the end of each stage whereas maximal heart rate and breath-by-breath respiratory parameters (Jäger, Germany) were simultaneously recorded (not reported in this study). The highest VO<sub>2</sub> averaged over 30 sec was taken as peak oxygen uptake.

**Main experimental procedure.** On arriving at the laboratory the electro cap (QuickCap, Compumedics, USA) was attached to the participant's head. A relaxing period where they lay supine for 10 min was followed by seating all subjects on the training machine M3 (Schnell, Germany) with a hip flexion of 110° and the knee flexion at 90°. The rotation axis of the dynamometer was visually aligned in relation to the lateral femoral condyle. The pad was removed from the lever arm to ensure a direct and accurate reaction of the electrogoniometer. To avoid pain sensations the subjects wore shin pads. Thereafter the signals were tested for quality (EEG: visually and impedance test), an EEG measurement was conducted at rest for two minutes (1' eyes open and 1' eyes closed, not reported in this paper). The experiment consisted of a sensorimotor task and an exercise protocol.

Sensorimotor function was tested by measuring the accuracy in an active knee angle reproduction task (Baumeister et al. 2008a) before (PRE), directly after (POST) and 60 min (REC) after the prolonged exhaustive exercise protocol. The subjects were asked to reproduce a given knee angle of 40° by moving the lever arm (without any additional weight) of the dynamometer from 90° to the target angle (full extension was equivalent to 0°). During three practice trials visual feedback (represented by a red horizontal line on a video monitor) was given while the target angle should be reached and memorized as accurately as possible. After that the visual feedback was taken away and the subjects were asked to repeatedly reproduce the target angle for ten times at their individual pace.

After the sensorimotor task MVC was determined at the best of three maximal voluntary isometric contractions (5sec with 2 min rest between) with the knee extensors. The subject's position – still seated on the M3 training machine in a standardized position with their knees flexed 90° – had been secured with waist and thigh straps.

During the exercise protocol the subjects were required to cycle at 70% of their individual  $VO_{2peak}$  for an hour in the laboratory followed by an incremental test. The test starts at the next lower 50 watts step and the regime followed the protocol performed in the preliminary testing to ensure exhaustion.

**Verifying fatigue (MVC).** The state of fatigue following the exercise protocol (POST) was verified by changes in peak torque in the MVC test compared to the PRE testing. A fatigue index was calculated according to the formula (Ribeiro et al. 2007):

$$\text{Fatigue}_{\text{index}} [\%] = (\text{PRE}_{\text{peak torque}} - \text{POST}_{\text{peak torque}}) / \text{PRE}_{\text{peak torque}} \times 100 \quad (1)$$

**Sensorimotor performance.** An electro-goniometer attached to the training machine M3 measured of lever arm motion. The error in reproduction accuracy is calculated as the absolute aberration between the target angle with and without visual feedback. Errors were averaged over the trial (Baumeister et al. 2008a) in each condition.

**EEG data acquisition.** EEG activity was recorded continuously using 22 Ag/AgCl electrodes (F7, F3, Fz, F4, F8, FC3, FCZ, FC4, C3, CZ, C4, CP3, CPZ, CP4, T5, P3, PZ, P4, T6, O1, OZ, O2) embedded in an elastic cap (QuickCap™, Compumedics Neuroscan, USA) in accordance with the international 10:20 system. All EEG data were recorded and stored using the NuAmps amplifier system (Compumedics Neuroscan, USA). A midforehead placement of the ground electrode and linked earlobes as averaged reference  $((A1+A2)/2)$  (Nunez and Srinivasan 2006) was used. The signals were sampled at 1000 Hz / 32 bit and amplified. Recordings were highpass filtered at 0.5 Hz and lowpass filtered at 100 Hz. An impedance test ( $< 5 \text{ k}\Omega$ ) ensured a sufficient signal to noise ratio before each measurement. For analysis the physiological signals were bandpass filtered (0.5 – 40 Hz). The EEG recordings were visually inspected by experienced electroencephalographers for artifacts. Only artifact free segments were used for analysis.

**EEG data analysis.** In the baseline-corrected EEG signals, Fast Fourier Transformation (FFT) was calculated over  $\approx 2$  s epochs (2048 sample Cosine windows) for all artifact free segments with the SCAN Software (SCAN 4.3; Compumedics Neuroscan, USA). Only data sets including at least 20 epochs were taken into account. Power spectra were calculated for each epoch and averaged (using a cosine window/10% extent of taper). Frequency domain

data were then imported to and analysed in MATLAB (The Mathworks Inc., USA). The power spectra were divided into different frequencies: Theta (4.75 – 6.75 Hz), Alpha-1 (7.0 – 9.5 Hz) and Alpha-2 (9.75-12.5 Hz). For statistical analysis the average power values were log-transformed (Pivik et al. 1993). Spectral power in the present frequency bands were computed across the three measurements (PRE, POST, REC) in each subject.

**Reliability of measures.** In preliminary methodological investigation test-retest measures of sensorimotor function and EEG spectral power had been demonstrated to be reliable. Healthy male individuals ( $24.2 \pm 3.1$  years;  $n=20$ ) were examined in a week to week design. For the performance of the JPS task the intraclass correlation coefficient (ICC) was 0.64. The EEG spectral power was divided into the different frequencies which demonstrated reliable ICC's for all 28 electrode positions [(lowest ICC (electrode position) – highest ICC (electrode position)); Theta: 0.87 (Oz) - 0.95 (Fz); Alpha-1: 0.89 (T3) – 0.97 (F4); Alpha-2: 0.90 (TP7) – 0.97 (Pz)]

**Statistical analysis.** For statistical analysis SPSS 17.0G software was used. All results are given as mean values and standard deviation. The Kolmogorov-Smirnov (K-S) test was used to determine if variables fit the Gaussian distribution. To examine sensorimotor function and cortical activity in different frequencies an ANOVA with repeated measures with factor CONDITION (PRE, POST, REC) was conducted. Compound symmetry, or sphericity, was verified by the Mauchly test. When the assumption of sphericity was not met, the significance of F-ratios was adjusted according to the Greenhouse-Geisser procedure. If ANOVA demonstrate a significant main effect for CONDITION, pairwise t-tests were made post hoc using Bonferroni adjustments in order to control for Type I error. The outcome of statistical calculations were declared significant if  $p \leq 0.05$ . Partial eta squared ( $\eta_p^2$ ) were computed to measure the effect sizes, which ranged from small (0.01 – 0.05) to medium (0.06 – 0.13) to large ( $\geq 0.14$ ). In case of significances in post hoc tests effect sizes were calculated according to Cohen's d. Here an effect size of 0.20 implied a small effect, 0.50 a medium effect, and 0.80 a large effect.

## RESULTS

**Verifying fatigue.** All participants demonstrate a decrease in MVC peak torque from PRE to POST. The fatigue index [%] demonstrate a decline in MVC performance of  $M=26.63$ ,  $SD=10.87$ .

**Sensorimotor performance.** Due to the exercise protocol there was a main effect in performance demonstrated by differences in reproduction error [°] ( $F_{2,22}=5.762$ ,  $p=.010$ ,  $\eta_p^2=.344$ ; FIGURE 1) during the JPS task in different conditions. Post-hoc analysis demonstrate a significant higher error directly after the exhaustive exercise (POST:  $M=6.74$ ,  $SD=4.32$ ) compared to PRE ( $M=3.14$ ,  $SD=1.55$ ,  $T(11)=2.547$ ,  $p=0.27$ ,  $d=0.87$ ) and lower values at REC ( $M=3.41$ ,  $SD=3.18$ ) compared to POST ( $T(11)=3.133$ ,  $p=0.01$ ,  $d=0.94$ ).

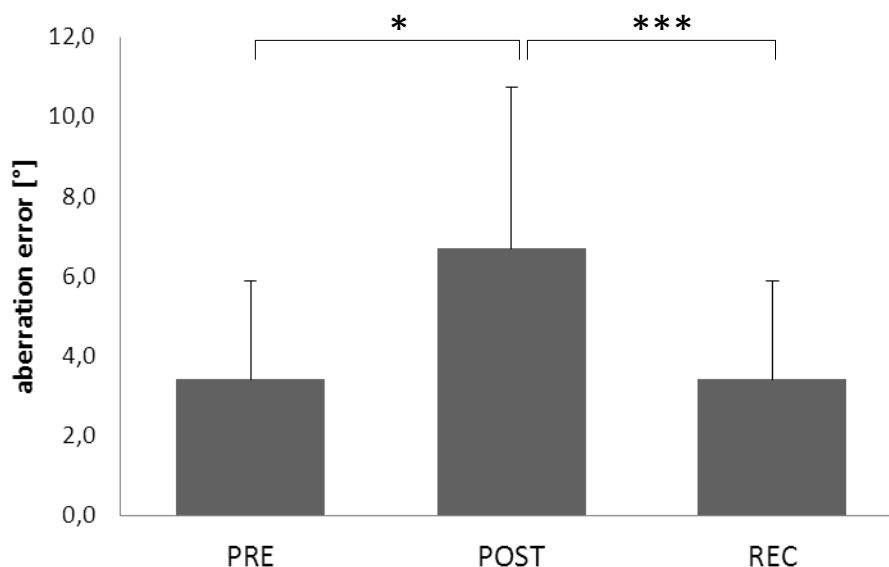


FIGURE 1. Accuracy in a joint position sense (JPS) task as a measure of sensorimotor function before, directly after and 60 min after exhaustive exercise (\*  $p\leq 0.05$ ; \*\*  $p\leq 0.01$ ; \*\*\*  $p\leq 0.001$ )

**Brain activity.** The EEG data of all subjects ( $n=12$ ) were included in the analysis with a minimum of 20 epochs and a mean epoch number of 27. The statistical analysis revealed significant differences in all frequency bands (Theta, Alpha-1, Alpha-2; FIGURE 2). If there is



a main effect it will be described in the following section completed by minimum and maximum values [ $\log \mu V^2$ ] in each frequency. All post hoc results related to the significant main effects are demonstrated in TABLE 2.

The maximum spectral power value in the Theta frequency band in PRE condition was detected at frontal brain areas (Fz: M=2.69, SD=0.52) whereas the lowest value could be seen at occipital electrode positions (Oz: M=1.75, SD=0.32). The statistical analysis demonstrated a significant CONDITION effect at frontal (F3:  $F_{2,22}=17.742$ ;  $p \leq .001$ ;  $\eta_p^2=.617$ ; Fz:  $F_{2,22}=13.654$ ;  $p \leq .001$ ;  $\eta_p^2=.577$ ; F4:  $F_{2,22}=21.587$ ;  $p \leq .001$ ;  $\eta_p^2=.665$ ; FIGURE 3) and fronto-central brain areas (FC3:  $F_{1,272,13,987}=17.352$ ;  $p \leq .001$ ;  $\eta_p^2=.612$ ; FCz:  $F_{1,283,14,118}=15.337$ ;  $p \leq .001$ ;  $\eta_p^2=.582$ ; FC4:  $F_{2,22}=15.271$ ;  $p \leq .001$ ;  $\eta_p^2=.604$ ).

TABLE 2 Overview post hoc results of significant CONDITION effects in Theta, Alpha-1 and Alpha-2 frequency bands

POS	THETA		ALPHA-1		ALPHA-2	
	PRE - POST	POST-REC	PRE -POST	POST-REC	PRE -POST	POST-REC
F7						
F3	↓ *** (1.60)	↑ *** (1.39)	↓ * (1.04)			↑ *** (1.95)
Fz	↓ *** (1.22)	↑ *** (2.17)	↓ * (0.94)			↑ ** (1.26)
F4	↓ *** (1.70)	↑ *** (2.77)	↓ ** (1.12)		↑ * (0.91)	↑ *** (1.53)
F8						
FC3	↓ *** (1.52)	↑ *** (2.43)	↓ * (0.98)			↑ ** (1.78)
FCz	↓ *** (1.44)	↑ *** (2.13)				↑ ** (1.41)
FC4	↓ ** (1.45)	↑ *** (2.40)	↓ * (1.42)		↑ * (1.03)	↑ ** (1.69)
C3						
Cz						↑ * (1.13)
C4						↑ * (1.16)
CP3						↑ * (1.19)
CPz						
CP4						↑ * (1.27)
T5			↓ ** (1.70)	↓ ** (1.49)	↓ ** (1.34)	↑ ** (1.67)
P3					↓ ** (1.14)	↑ * (1.18)
Pz						↑ * (1.31)
P4			↓ * (0.90)			↑ ** (1.63)
T6						↑ ** (1.34)
O1			↓ * (1.02)			↑ * (1.31)
Oz			↓ * (1.06)			↑ ** (1.74)
O2			↓ *** (1.84)			↑ ** (1.78)

↑ = increased EEG power, ↓ decreased EEG power; Bonferroni corrected level of significance: \*\*\*  $\leq 0.0003$ , \*\*  $\leq 0.003$ , \*  $\leq 0.017$ ; (Cohen's d: calculated for *within-subjects* data based on mean1/SD, mean2/SD and correlation between means);

ANOVA detected a significant main effect in Alpha-1 frequency spectral power (PRE maximum FCz: M=2.51, SD=0.64; minimum F7: M=1.77,SD=0.69) all over the scalp at frontal (F3, Fz, F4:  $F_{2,22}=6.543-14.834$ ;  $p\leq.011$ ;  $\eta_p^2\geq.409$ ), fronto-central (FC3, FCz, FC4:  $F_{2,22}=7.797-10.847$ ;  $p\leq.008$ ;  $\eta_p^2\geq.415$ ), central (Cz, C4:  $F_{2,22}=5.254-8.817$ ;  $p\leq.014$ ;  $\eta_p^2\geq.323$ ), centro-parietal (CP3, CP4:  $F_{2,22}=4.383-8.175$ ;  $p\leq.026$ ;  $\eta_p^2\geq.305$ ), parietal (P3, Pz, P4:  $F_{2,22}=3.533-7.541$ ;  $p\leq.047$ ;  $\eta_p^2\geq.243$ ), temporal (T5, T6:  $F_{2,22}=6.468-12.853$ ;  $p\leq.006$ ;  $\eta_p^2\geq.370$ ) and occipital (O1, Oz, O2:  $F_{2,22}=6.278-6.904$ ;  $p\leq.010$ ;  $\eta_p^2\geq.386$ ) brain areas.

Spectral power in Alpha-2 frequency (PRE maximum Pz: M=2.89, SD=0.89; minimum T5 M=2.07, SD=0.65) demonstrated a significant main effect due to CONDITION at F4 ( $F_{2,22}=6.276$ ;  $p=.007$ ;  $\eta_p^2=.363$ ), FC4 ( $F_{2,22}=5.960$ ;  $p=.009$ ;  $\eta_p^2=.373$ ), T5 ( $F_{2,22}=14.379$ ;  $p\leq.001$ ;  $\eta_p^2=.590$ ) and P3 ( $F_{2,22}=5.416$ ;  $p=.012$ ;  $\eta_p^2=.330$ ).

## DISCUSSION

Prolonged exhaustive exercise leads to significantly decreased accuracy in a knee angle matching task and was accompanied by an altered brain state demonstrated in specific changes in frontal Theta, overall Alpha-1 and parietal Alpha-2 spectral power due to the different conditions before, directly after and 60 minutes after inducing fatigue.

### Sensorimotor performance

Knee angle matching tasks were widely used to evaluate sensorimotor control. It was hypothesized that prolonged exhaustive exercise on a cycle ergometer has an effect on precision in motor behavior. The current results clearly demonstrate that fatigue as a consequence of exhaustive exercise lead to decreased accuracy in active knee angle reproduction. Proprioceptive feedback with the muscle spindles primarily responsible for joint position sensation seems to be affected by fatigue. It has been discussed that the modification of the afferent proprioceptive information may be altered by an increased threshold of muscle spindle discharge (Pedersen et al. 1999).

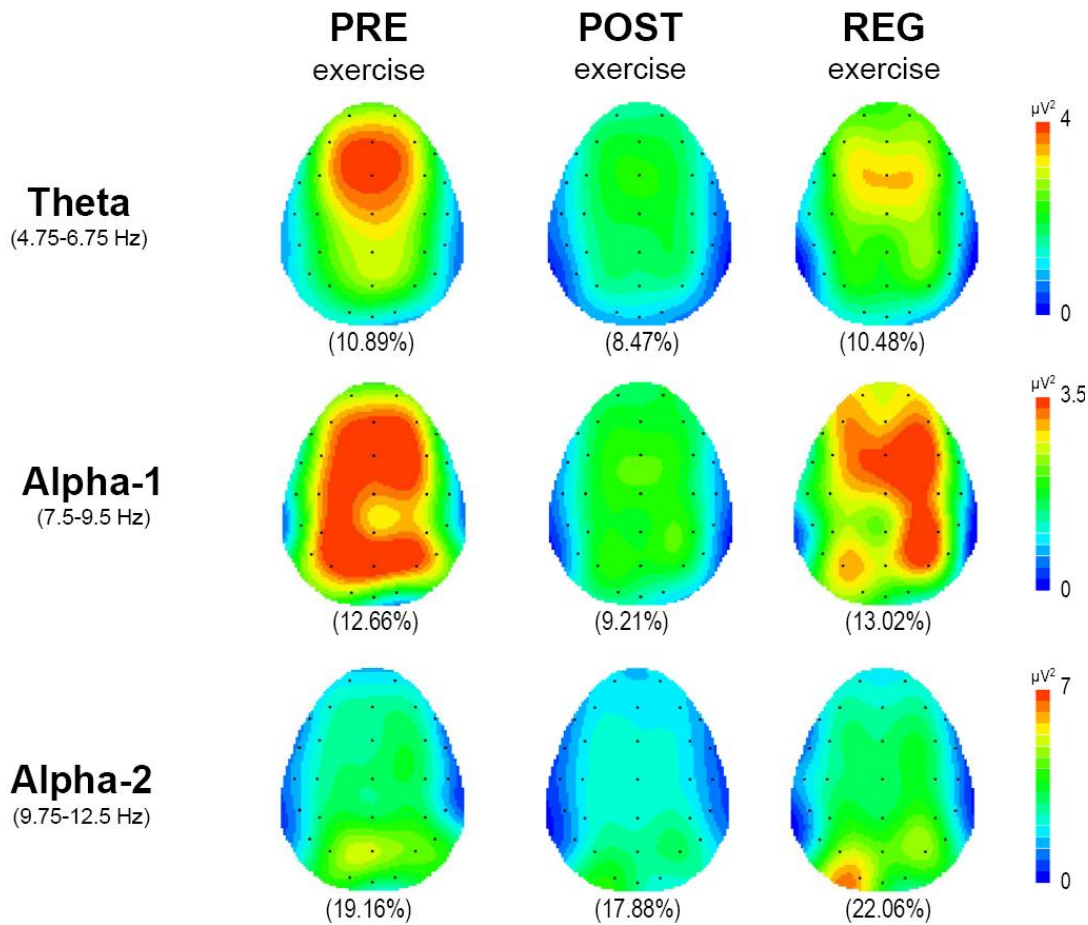


FIGURE 2. Topography of EEG power over the scalp average in Theta, Alpha-1, Alpha-2 and Beta-1 frequency bands in different conditions averaged over all participants (n=12)

A number of studies support these results. In the initial study to investigate sensorimotor control and fatigue Skinner et al. (1986) detected a significant decrease in the subject's ability to reproduce knee joint angles actively after a sports related fatigue protocol (3.75 miles track and field running followed by two treadmill sessions). These findings were supported by the work of Lattanzio et al. (1997) who used three different cycle ergometer fatigue protocols (ramp test to exhaustion, 80%  $VO_{max}^2$  interval), each standardized to participants fitness level, and demonstrated a significant increase in knee position matching error after all three protocols. Furthermore Givoni et al. (2007) demonstrated significant effects with an increased reproduction error after concentric as well as eccentric induced fatigue in a knee angle reproduction task. The same results have been shown by Ribeiro et

al. (2007) who used a concentric isokinetic exercise protocol to induce fatigue in an elderly population.

All experimental results were explained with deficits in knee angle matching accuracy as a result of an impaired peripheral mechanism with little attention paid to the CNS, but they also propose the need to address the role of the brain in this process.

### Brain activity.

The main purpose of the present study was to characterize changes in cortical activity in sensorimotor functioning which are related to the fatigue conditions before (PRE), directly after (POST) and after 60 min of recovery (REC). The observations revealed systematic effects in Theta, Alpha-1 and Alpha-2 frequency bands which will be discussed related to sensorimotor control in the following paragraphs.

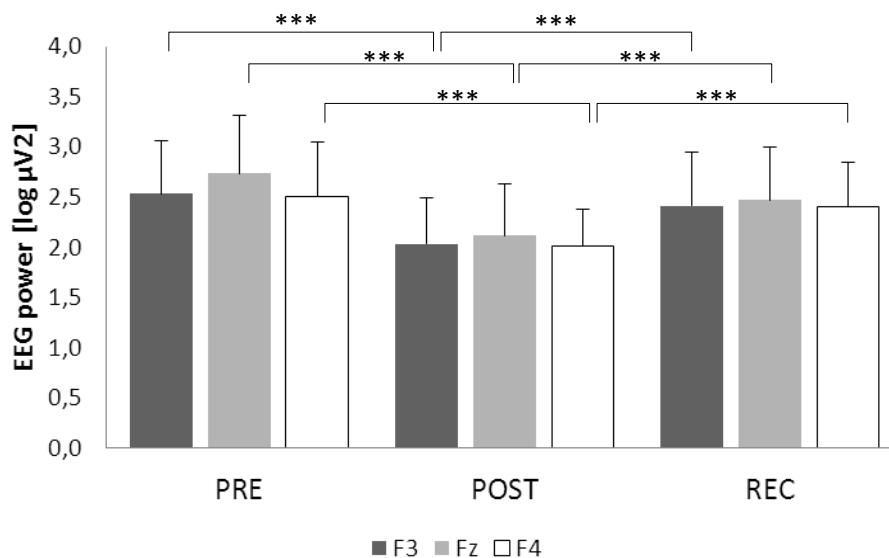


FIGURE 3. Theta EEG power values at frontal brain areas (F3, Fz, F4) before, directly after and 60 min after prolonged exhaustive exercise (\*  $p \leq 0.05$ ; \*\*  $p \leq 0.01$ ; \*\*\*  $p \leq 0.001$ ).

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### *Theta Frequency (4.75-6.75 Hz)*

The most prominent finding was a significant Theta spectral power decrease which was found in frontal areas of the brain (see FIGURE 2 & 3). The effect recovers after 60 min demonstrated by a significant increase of Theta spectral power values in the same area.

The Theta frequency is an EEG signal in the range from around 4-7 Hz described as prominent in frontal regions of the cortex in cognitive (Smith et al. 1999; Jensen & Tesche 2002), sensorimotor (Slobounov et al 2001; Grunwald et al. 2002; Baumeister et al. 2008a; Baumeister et al 2011) and complex sports related tasks (Doppelmayr et al 2008; Baumeister et al. 2008b).

Much research in recent years has focused on human frontal Theta oscillations related to attentional resources and neurocognitive demands (Klimesch 1997; Gevins et al. 1997; Sauseng et al. 2007) There is evidence from combined fMRI-EEG (e.g. Gevins et al. 2007) and sLORETA (e.g. Doppelmayr et al. 2008) approaches that the frontal Theta signal is generated in areas of the anterior cingulate cortex (ACC) which is thought to be part of the human executive attentional system involved in mechanisms for monitoring and modulating sensory input (Posner & Rothbart 2007).

The decrease in spectral Theta power values after prolonged exhaustive exercise must have to be discussed as a decrease in executive attention which causes problems in monitoring and modulating sensory inputs. With decreased attentional resources it is difficult to maintain precise sensorimotor control. The higher aberration error directly after the prolonged exhaustive exercise protocol supports that argument. One hour after the exhaustive exercise the performance as well as frontal Theta power recover and might demonstrate a time dependency of this process.

### *Alpha Frequency (7.0-12.5 Hz)*

Changes in the Alpha frequency band also demonstrate a characteristic pattern due to the prolonged exhaustive exercise protocol. Alpha is the dominant frequency in the human EEG of adults and is thought to be generated in widespread areas of the cortex through cortico-

cortical and thalamo-cortical interactions (Niedermeyer and Lopes da Silva 2005). The Alpha frequency is divided into two subgroups: a lower (Alpha-1, 7.0-9.5 Hz) and an upper (Alpha-2, 9.75-12.5 Hz) component. The lower alpha-1 component is attenuated over broad regions of the cortex and is thought to play a role in alerting mechanisms and expectancy processes (Smith et al. 1999). Alpha-1 activity is often described as a form of cortical idling with its amplitude inversely related to the number of neuronal populations activated during cognitive and sensorimotor processes (Gevins et al. 1997; Niedermeyer and Lopes da Silva 2005). In a fatigued state the results demonstrated a decrease in Alpha-1 spectral power widespread over the brain whereas sixty minutes after the exhaustive exercise the Alpha-1 power increased again. Therefore a suppression of the Alpha frequency, interpreted as 'desynchronisation', suggested that a large population of neurons no longer oscillate in synchrony due to information processing (Klimesch 1997). It can be argued that the activation after the exercise protocol is due to an increased alertness which may be caused by incoming sensory stimuli due to the fatigue condition.

It is widely accepted that the Alpha-2 component tends to be attenuated by task-specific demands depending on parieto-occipital regions in the area of the somatosensory cortex (Gevins et al. 1997; Smith et al. 1999; Neuper et al. 2006). Following the same inverse relationship between power and neuronal activation the increased Alpha-2 parietal power before the exhaustive exercise implies less cortical activation in the somatosensory areas compared to the fatigue condition directly after. Sixty minutes after the end of exercise Alpha-2 band power recovers back to PRE fatigue values. Ribeiro et al. (2007) theorized that fatigue impairs the proprioceptive and kinaesthetic properties of joints by changing the threshold of muscle spindle discharge. Consequently that may lead to an altered afferent feedback (Pedersen et al. 1999) which requires more neuronal resources for processing this changed sensory information in somatosensory areas. In this context Pfurtscheller et al. (2000) and Neuper et al. (2006) argue that desynchronization during motor tasks in the parietal upper Alpha frequency band indicate enabled information processing in this brain areas. It can be assumed that the changed afferent feedback due to the fatigue protocol

cause an increased information processing in the somatosensory areas to react to the changed afferent feedback.

Overall, the electrophysiological observations in this pilot study were able to describe specific characteristics in brain activation pattern due to induced fatigue and resulting in decreased precision in motor behavior. In detail the results demonstrate (1) a decrease in frontal theta power, (2) a decrease in overall Alpha-1 power and (3) a decrease of parietal Alpha-2 power. Furthermore the results indicate that these effects recover 60 min after induced fatigue. Future studies have to look closer into the different patterns related to sensorimotor control in fatigue conditions. It may be helpful to elucidate the role of fatigue using phasic changes in power values (ERD/ERS) in sensorimotor control (Neuper et al.2006), cortico-cortical and cortico muscular coherence as a measure of connectivity between brain areas and muscles as well as to apply source localization procedures. Based on the observations in this study and future results from different proposed EEG approaches it will be possible to propose a detailed research model of the influence of different fatigue conditions on sensorimotor control.

## **CONCLUSION**

The observations of this study were able to add information to the influence of fatigue on sensorimotor control. Exhaustive exercise affects brain pattern demonstrated by changes in electro-cortical activations in Theta and Alpha frequency bands. These changes are accompanied by decreased precision in a sensorimotor knee angle reproduction task. After a sixty minute recovery period task performance increased again and EEG power get nearly back to baseline values. Further research is needed to validate the observations of this study and to elucidate brain mechanism in fatigue conditions in more detail. The knowledge of this mechanisms may have a high impact on injury risk management, prevention strategies and optimizing performance in sports and exercise.

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## PUBLICATION 2

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<b><i>Acquisition of data:</i></b>	<b>von Detten, van Niekerk, Schubert</b>
<b><i>Analysis and interpretation</i></b>	<b>Baumeister, von Detten, Schubert</b>
<b><i>Drafting the manuscript</i></b>	<b>Baumeister</b>
<b><i>Critical intellectual revision</i></b>	<b>Louw, Ageberg</b>
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## ABSTRACT

Landing from a jump is related to predictive sensorimotor control. Frontal, central and parietal brain areas are known to play a role in this process based on online sensory feedback. This can be measured by EEG. However, there is only limited knowledge about brain activity during predictive preparation of drop landings (DL). The purpose is to demonstrate changes in brain activity in preparation of DL in different conditions. After resting, 10 athletes performed a series of DL's and were asked to concentrate on the landing preparation for ten seconds before an auditory signal required them to drop land from a 30cm platform. This task was executed before and after a standardized fatigue protocol. EEG spectral power was calculated during DL preparation. Frontal Theta power was increased during preparation compared to rest. Parietal Alpha-2 power demonstrated higher values in preparation after fatigue condition while lower limb kinematics remained unchanged. Cortical activity in frontal and parietal brain areas is sensitive for predictive sensorimotor control of drop landings. Frontal Theta power demonstrates an increase and is related to higher attentional control. In a fatigued condition the parietal Alpha-2 power increase which might be related to a deactivation in the somatosensory brain areas.

**Key words:** motor, fatigue, preparation, cortical activity

## INTRODUCTION

Landing from a jump in sports and exercise is one of the prominent noncontact mechanisms for ankle and knee injuries. It presents a significant challenge to athletes' motor control prior to and during contact with the supporting surface. Even if some laboratory based injury prevention programs had been able to change the movement pattern of landing maneuvers and reduce the potential risk of injury [14,5], there are still conflicting results in the effect of such programs on lower limb injury prevention [6]. Biomechanical data shed light on the evaluation of intervention programs, but cannot elucidate the underlying physiological mechanism, on which the program should be based. Consequently, there is a need for more insight into the sensorimotor control process behind the biomechanically described motor output of drop landings [3].

Successful landing is based on sensorimotor control, a dynamic interaction between sensation of sensory information, the integrating of information in the central nervous system and motor output to perform voluntary movements and postural control. The electroencephalography (EEG) can be used to measure brain activity during the performance of sensorimotor tasks and has already proved to be able to describe information processing during sensorimotor control [25,1]. One prominent concept of information processing which is involved in sensorimotor control is the working memory [27,23]. This concept is defined as a limited capacity system which is involved in control, regulation and active maintenance of task-relevant information and links long-term memory, perception and action [23]. Working memory processes do not rely on a single anatomical location, but has to be considered a network most likely located in frontal and parietal brain areas [26].

Sensorimotor tasks had been used to demonstrate the sensitivity of frontal, central and parietal brain areas in Theta and Alpha frequencies [27,11,25,7,1,2]. In more detail frontal Theta values increase with higher demand of attentional control and information processing demand whereas the slow alpha band component (Alpha-1) is attenuated over broad regions of the cortex and is thought to be a non-specific attentional and expectancy process indicator. The fast Alpha (Alpha-2) values are prominent in parietal brain areas and

decrease, because of an inverse relationship between Alpha power and the activation of neuronal populations in the underlying somatosensory area [16].

Sensorimotor control in the above process relies on the principle of online sensory feedback sensation. But an important part of a drop landing task is the preparation which is planned in advance and not altered by online peripheral sensory feedback prior to ground contact [28]. The CNS accurately prepares the muscles for self-initiated landings most likely based on an internal model of the dynamics of the limbs [28,12]. Instead of online sensations from peripheral receptors, calculated sensory information from this model is predictively integrated into the movement preparation and processed at the brain level.

To our knowledge the involved brain areas in this predictive sensorimotor control of drop landings had not yet been studied. In this first pilot study of a series of experiments, we have included healthy participants. The aim of this pilot study is to demonstrate changes of EEG measures in predictive preparation of drop landings due to different conditions (rest, before and after a fatigue protocol) in frontal and parietal brain areas. We expect to detect activity changes in frontal Theta and parietal Alpha frequencies which is comparable to sensorimotor feedback control. This will provide the methodological basis for the detection of altered brain activity as a possible mechanism for lower extremity injury (e.g. ankle, knee).

## **METHODS**

**Participants.** Ten healthy, right-handed athletes (7 males/3 females;  $21.1 \pm 1.6$  years;  $72.7 \pm 12.2$  kg;  $177 \pm 12$  cm) volunteered to participate in the study and completed all study requirements (no lower limb injuries 6 month prior to data collection, no surgery to the lower limbs and no subjects suffering from overtraining syndrome). All subjects were competitive athletes from the following sports: soccer (3), rugby (7) and netball (1) and had a Tegner activity score [28] of 9 ( $n=8$ ) or 10 ( $n=2$ ) at the time of the experiment. The participants had no history of neurological, cardiovascular or other major disorders and did not use any medications or drugs at the time of assessment. They were asked to have no physical or

psychological exposures (e.g. intensive training sessions, examinations) 24 hours prior to the exercise trials. Written informed consent was obtained from each participant, and the experimental procedures were approved by the local ethics committee of the Stellenbosch University, South Africa [N11/01/017] and meet the ethical standards of the International Journal of Sports Medicine [10].

**Experimental procedure.** On arriving at the laboratory the EEG electro cap (QuickCap) was attached to the participants head and the reflective markers were placed on bony landmarks of the lower limb. After 10 minutes relaxing the signals were tested for quality (EEG: visually and impedance test) and an EEG measurement was conducted at rest (REST). The athletes were asked to perform a series of six successful drop landings without any changes in instructions or conditions. The subjects stood on a 30cm platform and were instructed to mentally prepare for the drop landing for ten seconds before an auditory signal required the subjects to drop off the platform with arms held at the sides and land with both feet on the floor. After they finished the drop landing series (PRE) all participants carried out a standardized fatigue protocol which was followed by a DL series again in a fatigued state (POST).

**Fatigue protocol.** After the pre fatigue trials (M1) the maximum jumping height (100%) of each subject was measured and marked. The participants were asked to perform a functional fatigue protocol. The protocol consists of performing approx. up to 8 sets of 15 vertical jumps (30 sec rest between sets). Subjects were deemed to be fatigued when they were not able to reach 80% of their maximum jump height in three consecutive jumps. An additional test jump after the protocol was measured to quantify the fatigue index [3]:

$$\text{Fatigue}_{\text{index}} [\%] = (\text{PRE}_{\text{jump height}} - \text{POST}_{\text{jump height}}) / \text{PRE}_{\text{jump height}} \times 100 \quad (1)$$

**EEG data acquisition and analysis.** EEG activity was recorded continuously using Ag/AgCl electrodes (F7, F3, Fz, C3, CZ, C4, P3, PZ, P4) embedded in an EEG cap (QuickCap™, Compumedics Neuroscan, USA) in accordance with the international 10:20 system. All EEG data were recorded and stored using the NuAmps amplifier system (Compumedics

Neuroscan, USA). A midforehead placement of the ground electrode and linked earlobes as averaged reference  $((A1+A2)/2)$  [17] was used. The signals were sampled at 1000 Hz / 32 bit and amplified. Recordings were highpass filtered at 0.5 Hz and lowpass filtered at 100 Hz. An impedance test ( $< 5 \text{ k}\Omega$ ) ensured a sufficient signal to noise ratio before each measurement. For analysis the physiological signals were bandpass filtered (0.5 – 40 Hz). A criterion for automatic artifact rejection was  $100 \mu\text{V}$  for EEG's. The electrooculogram (EOG) was recorded and used for ocular artifact rejection [24] provided by Scan 4.5 (Compumedics Neuroscan, USA). Then the epochs with minor artifacts were visually checked by experienced electroencephalographers and removed from further analysis. Only artifact free segments were used for analysis. In the baseline-corrected EEG signals, Fast Fourier Transformation (FFT) was calculated over  $\approx 2$  s epochs (2048 sample Cosine windows) for all artifact free segments with the SCAN Software (SCAN 4.4; Compumedics Neuroscan, USA). Only data sets including at least 20 epochs were taken into account. In all conditions a mean of 3 out of 30 sweeps were rejected from further analysis. To reduce the number of variables [18] we clustered 12 EEG channels into four topographical regions of interest: (1) mid-frontal (F3, Fz, F4), (2) central (C3, Cz, C4) and (3) mid-parietal (P3, Pz, P4).

Power spectra were calculated for each epoch and averaged (using a cosine window/10% extent of taper). Frequency domain data were then imported to and analysed in MATLAB (The Mathworks Inc., USA). The power spectra were divided into different frequencies: Theta (4.75 – 6.75 Hz), Alpha-1 (7.0 – 9.5 Hz) and Alpha-2 (9.75-12.5 Hz). For statistical analysis the average power values were log-transformed [21]. Spectral power in the present frequency bands were computed for the resting condition (M0) and the preparation period of each drop landing before and after the fatigue protocol (M1, M2) in each subject.

**Vicon Motion acquisition and analysis.** An eight Vicon T-series motion analysis system (Vicon Motion Systems (Ltd) (Oxford, UK) was utilised to analyse the hip, knee and ankle kinematic data. Integrated Vicon Software, Nexus 1.4 software, and giganet were used to process the data. Thirty-five retro-reflective markers (14 mm diameter) which reflect infrared rays were placed on ten bony landmarks of the lower limb according to the body Plug-in-Gait

model. The markers were placed on the anterior superior iliac spines, posterior superior iliac spines, lateral epicondyle of the femur, lateral thigh, lower third of the tibia, ankle, second metatarsal head and calcaneus. After all the trials were captured, each trial was reconstructed and markers were labelled according to the bony landmarks in the Nexus software. Data reduction was done in MATLAB (The Mathworks, Natick, MA). Three-dimensional angles at initial contact (IC) of the hip, knee and ankle were extracted. An IC event was calculated for both feet and the one with the earliest event was chosen. Angles of hip, knee and ankle in the sagittal plane at IC were calculated to control the motor output.

**Statistical analysis.** For statistical analysis SPSS 17.0G software was used. No primary outcome measure was determined, since the study has an exploratory character. We expect cortical changes of EEG power values (Theta, Alpha-1, Alpha-2) in frontal and/or parietal brain areas. Mean difference and 95% confidence intervals (95% CI) in cortical changes between rest and the preparation period (REST vs. PRE) as well as cortical and kinematic differences (hip, knee and ankle angles at IC for right and left limb) in fatigue related conditions (PRE vs. POST) were described and paired t-tests were used for comparisons. The Shapiro-Wilks test was used due to the small sample size and demonstrated statistic values  $> 0.90$  which was classified as normally distributed. The outcome of statistical calculations were declared significant if  $p \leq 0.05$ . If brain areas demonstrate a significant result, mean differences (95% CI) and paired t-tests were additionally calculated for each included electrode position (frontal=F3, Fz, F4; central=C3, Cz, C4; parietal=P3, Pz, P4).

## RESULTS

### *Brain activity in rest vs. preparation (REST vs. PRE)*

Significant differences were seen between resting (REST) and preparation (PRE) in different brain areas in Theta frequency [ $\log \mu V^2$ ] (Fig.1). The frontal area demonstrated the highest values in Theta power compared to the other cortical areas (Tab.1). The preparation period show increased power in frontal and parietal brain areas compared to the resting condition



(Tab.1). Significant increases could be seen in fronto-central position (Fz) from rest (M=2.82, SE=0.11) to preparation (M=3.09, SE=0.10; mean difference 0.27, 95% confidence interval 0.10 to 0.43;  $p=0.008$ ), whereas left frontal Theta (F3) tended to increase from rest (mean=2.62, SE=0.17) to preparation (M=2.85, SE=0.09; 0.23, -0.01 to 0.43).

The increase of Theta power values from rest to preparation in parietal brain areas was based on significant higher power values in left-parietal (P3: M0 mean=2.44, SE=0.10; PRE mean=2.69, SE=0.08; 0.25, 0.06 to 0.44;  $p=0.016$ ) and right parietal (P4: REST mean=2.52, SE=0.09; PRE mean=2.71, SE=0.08; 0.17, 0.03 to 0.30;  $p=0.018$ ) positions.

Alpha-1 and Alpha-2 power demonstrated no significant changes in the preparation of drop landings.

TABLE 1. EEG power values in frontal, central and parietal brain areas during rest (REST) and preparation (PRE)

[log $\mu V^2$ ]	BRAIN ACTIVITY		
	REST Mean (SE)	PRE Mean (SE)	Mean diff (95% CI) (PRE minus REST)
<b>THETA</b>			
Frontal	2.65 (0.11)	2.93 (0.05)	0.27 (0.06, 0.49)
Central	2.53 (0.11)	2.67 (0.10)	<b>0.13 (-0.19, 0.37)</b>
Parietal	2.55 (0.10)	2.78 (0.08)	<b>0.22 (0.07, 0.38)</b>
<b>ALPHA-1</b>			
Frontal	2.46 (0.19)	2.63 (0.17)	0.09 (-0.23, 0.57)
Central	2.46 (0.21)	2.59 (0.13)	0.12 (-0.29, 0.54)
Parietal	2.60 (0.22)	2.61 (0.14)	<b>0.01 (-0.31, 0.32)</b>
<b>ALPHA-2</b>			
Frontal	2.41 (0.20)	2.55 (0.24)	0.14 (-0.19, 0.48)
Central	2.88 (0.27)	3.04 (0.22)	0.16 (-0.30, 0.63)
Parietal	3.04 (0.20)	3.24 (0.21)	<b>0.20 (-0.10, 0.53)</b>

### *Verifying fatigue*

The fatigue index [%] demonstrated a decrease in performance of mean=78.72 and SD=8.1 which fits the verification of fatigue.

TABLE 2. EEG power values in frontal, central and parietal brain areas before (PRE) and after (POST) a fatigue protocol

[log $\mu V^2$ ]	BRAIN ACTIVITY		
	PRE Mean (SE)	POST Mean (SE)	Mean diff (95% CI) (POST minus PRE)
<b>THETA</b>			
Frontal	2.93 (0.05)	2.77 (0.11)	-0.16 (-0.40, 0.08)
Central	2.67 (0.10)	2.65 (0.13)	-0.02 (-0.35, 0.22)
Parietal	2.78 (0.08)	2.75 (0.07)	-0.03 (-0.29, 0.24)
<b>ALPHA-1</b>			
Frontal	2.63 (0.17)	2.54 (0.20)	-0.09 (-0.21, 0.02)
Central	2.59 (0.13)	2.43 (0.14)	-0.15 (-0.35, 0.04)
Parietal	2.61 (0.14)	2.64 (0.16)	-0.03 (-0.24, 0.23)
<b>ALPHA-2</b>			
Frontal	2.55 (0.24)	2.70 (0.25)	0.15 (-0.05, 0.27)
Central	3.04 (0.22)	3.03 (0.19)	-0.01 (-0.15, 0.23)
Parietal	3.24 (0.21)	3.46 (0.21)	0.22 (0.06, 0.38)

*Brain activity in different conditions (PRE vs. POST)*

Theta and Alpha-1 power values demonstrated no significant changes (Tab.2), whereas Alpha-2 power show highest power values in parietal brain areas and increased significantly after the fatigue protocol (Fig.1; Tab.2). A closer look at the parietal electrode positions obtained significant increases at P3 (M1: mean=3.17, SE=0.22 and M2: mean=3.43, SE=0.21; 0.26, 0.08 to 0.44;  $p=0.010$ ) and Pz (M1: mean=3.30, SE=0.22 and M2: mean=3.55, SE=0.25; 0.25, 0.02 to 0.49;  $p=0.037$ ).

*Landing pattern at initial contact in different conditions (PRE1 vs. POST)*

The biomechanical joint angles of hip, knee and ankle in the sagittal plane demonstrated no differences related to the fatigue protocol which indicates the same movement pattern after the fatigue protocol (Tab.3).

TABLE 3. Kinematics of the lower limb at initial contact (IC) before (M1) and after (M2) fatigue protocol

<b>Joint angle</b> [°]	<b>PRE</b> Mean (SE)	<b>POST</b> Mean (SE)	<b>Mean diff (95% CI)</b> (M2 minus M1)
<b>Right limb</b>			
IC hip flex	35.0 (4.2)	34.4 (3.6)	-0.6 (-3.4, 2.2)
IC knee flex	41.6 (4.8)	40.8 (4.2)	-0.8 (-3.3, 1.6)
IC ankle DF	13.0 (1.2)	11.9 (1.4)	1.1 (-2.7, 0.3)
<b>Left limb</b>			
IC hip flex	36.6 (3.8)	37.3 (3.4)	0.7 (-1.7, 3.32)
IC knee flex	45.7 (4.4)	46.8 (3.9)	1.1 (-0.8, 2.9)
IC ankle DF	14.7 (1.0)	14.7 (1.2)	0.0 (-1.4, 1.4)

## DISCUSSION

This is the first study to illustrate the changes of EEG measures in predictive sensorimotor control during the preparation of a drop landing task under different conditions. The results in frontal Theta power from a resting situation to the preparation period and parietal Alpha-2 power before and after a fatigue protocol demonstrated increased EEG power values. Both locations and associated frequencies were thought to be part of the working memory concept [1,27,23], a limited capacity system which is involved in control, regulation and active maintenance of task-relevant information [13].

### *Brain activity in rest vs. preparation (REST vs. PRE)*

The preparation of a drop landing was associated with higher Theta power values in frontal brain areas compared to the resting state. Theta power is consistently described as prominent in frontal brain areas in sensorimotor [24,9,1] and complex sports-related tasks [7,2]. Research in the last decade has linked frontal Theta oscillations with the anterior

cingulate cortex (ACC) related to executive attentional control. Executive attention involves the control and regulation of neural activity related to task complexity, a function of information processing demands [2] and is thought to be part of the central executive of the working memory model [13]. It is well established by combined EEG-fMRI and cortical source analyzing (sLORETA) studies that the generation of frontal Theta frequency is located in the ACC [8,7]. Even if the role of the ACC is not fully understood, increased activation in this specific area has been observed during performance of tasks which require selection and/or inhibition of sensory information. Due to the “anterior attentional system” hypothesis [4,22] the ACC is thought to directly allocate attentional resources in response to complex information processing related to the central executive component of the working memory. Therefore, Theta power may serve as an indicator of neural activation in the ACC related to increased attention in frontal brain areas [27,11].

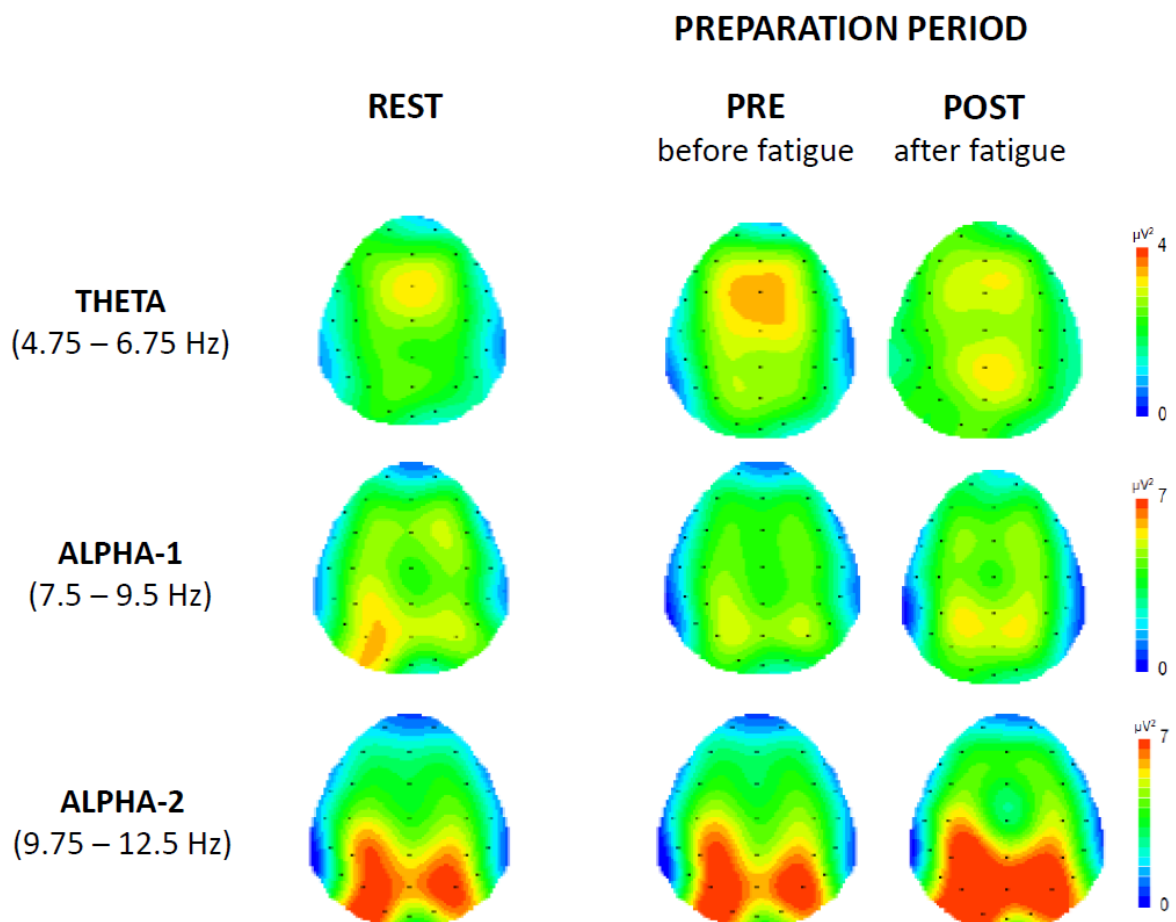


FIGURE 1. Topography of EEG power (grand average) over the scalp in rest and the preparation period before and after fatigue in Theta, Alpha-1 and Alpha-2 frequency bands over all participants (n=10)

In the current study the participants demonstrated higher frontal Theta power in the preparation period, which might reflect a higher attentional control for a successful landing. The predictive calculation and integration of information for landing (most likely from an internal model [12]) lead to a higher demand in processing which result in higher Theta values.

This model was first established for sensorimotor feedback control, where tasks rely on the principle of online sensory feedback sensation and processing. There is overwhelming evidence to support the sensitivity of frontal Theta power to sensorimotor feedback control. Smith et al. [26] showed an increase in frontal Theta with increased complexity during a sensorimotor control task (video game “frustrated maze”) due to time pressure. Increased frontal Theta power was also found by Baumeister et al. [1] at the frontal scalp positions in patients with anterior cruciate ligament injury (ACL) in a joint angle reproduction task. This finding is assumed to reflect changed afferent sensory information feedback due to receptor damage caused by the injury. In conclusion, the results of this study suggest that increased Theta can be observed during predictive sensorimotor control in the preparation for landing.

#### *Brain activity in different conditions (PRE vs. POST)*

A fatigue condition was used to change the information processing demand to investigate the sensitivity of frontal and parietal brain activity to different conditions. The results demonstrated no significant changes in Theta and Alpha-1, but higher Alpha-2 values in the preparation for landing after a fatigue protocol whereas the kinematics of the motor output remained unchanged during initial contact.

In the light of attentional control frontal Theta power demonstrated no significant changes after the fatigue protocol. It can thus be assumed that the information processing demand has not changed after the fatigue protocol.

This result is supported by a prominent power increase in the Alpha-2 frequency in parietal brain areas. It is well established that movement preparation and execution is characterized

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by a decrease in Alpha-2 frequency over the sensorimotor area (“desynchronization”; [19,15]). In contrast the current results show an increase of Alpha-2 power which can be found in the literature defined as “synchronization”. The term synchronization suggests that a population of neurons oscillate in synchrony and indicate a deactivation in the area of the somatosensory cortex. Pfurtscheller et al. [19] hypothesized that deactivated cortical areas may represent idling or inhibitory cortical activity, described with the ‘gating theory’ proposed by Neuper et al. [15]. The somatosensory cortex may not be engaged in the predictive preparation of drop landings and therefore may “close the gate” for incoming peripheral sensory information. In this case there would be no change in information processing demand which is supported by unchanged frontal Theta values in this condition. Future studies are needed to determine these results.

A few limitations must be taken into account. First, synchronization of Alpha frequencies is often described with the terms “habituation” or “neural efficiency” which means to gain the same outcome with less activity or effort. It is unlikely that the synchronization in Alpha-2 frequency in this study is due to one of these terms. Alpha-2 power increased above the resting level which seems to be due to an inhibition effect [15]. To overcome this limitation the next study should include a control group without a fatigue intervention to justify the effects. Second, the study demonstrated increased EEG power and 95% CI support that the observed differences are significant. Even if there is strong evidence of the relationship between frontal Theta and attention from other studies [8,7] it is only indirect in terms of attention or working memory control, because we have not directly measured electrophysiological values at the specific ACC location. Further studies should attempt to evaluate attentional control parameters to strengthen the relationship.

To our knowledge this was the first study to look into the cortical activity in the preparation period of drop landings. From our perspective the current pilot study added knowledge in the following areas:

The exploratory results demonstrate that cortical activity can be measured in predictive sensorimotor control and frontal and parietal brain areas are involved which fits the proposed working memory model.

Predictive sensorimotor control demonstrate an increase of Theta power in frontal brain areas during the preparation for landing, which may be related to attentional control and information processing demands

Induced fatigue in predictive sensorimotor control result in an Alpha-2 synchronization in parietal brain areas which may be related to a decrease or inhibition of information processing in the somatosensory cortex

The central approach using EEG measures adds valuable information about the preparation period of drop landings and expands biomechanical knowledge. From a methodological standpoint this study will provide a possibility for a detection of altered brain activity after ACL injuries well in line with recent research [1] and might help formulate verifiable hypothesis to have a more detailed look into predictive sensorimotor control. Only with knowledge of the sensorimotor control process and further research into the activity and communication of the involved brain areas related to biomechanical data we will be able to develop and evaluate effective injury prevention and also rehabilitation programs.

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## PUBLICATION 3

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<b><i>Analysis and interpretation</i></b>	<b>Baumeister, Barthel</b>
<b><i>Drafting the manuscript</i></b>	<b>Baumeister</b>
<b><i>Critical intellectual revision</i></b>	<b>Barthel, Geiß, Weiss</b>
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## ABSTRACT

The aim of the study was to investigate the effect of Phosphatidylserine (PS) on cognition and cortical activity after mental stress. After familiarization, 16 healthy subjects completed cognitive tasks after induced stress in a test –re-test design (T1 and T2). Directly after T1 subjects were assigned double-blind to either PS or placebo followed by T2 after 42 days. At T1 and T2 cortical activity was measured during baseline, and immediately after stress and the cognitive tasks using electroencephalography (EEG). EEG was recorded at 17 electrode positions and FFT determined power at Theta, Alpha-1, Alpha-2, Beta-1 and Beta-2. Statistics were calculated using ANOVA (group x trial x time). The main finding of the study was that chronic supplementation of phosphatidylserine significantly decreases Beta-1 power in right hemispheric frontal brain regions (F8,  $p < 0.05$ ) before and after induced stress. The results in Beta-1 power in the PS group were connected to a more relaxed state compared to the controls.

**Key words:** Phosphatidylserine, EEG, cortical activity, stress, cognitive tasks

## Introduction

Phosphatidylserine (PS) is an essential component of all biological membranes and has important regulatory functions within mammalian cells. In humans, PS is most concentrated in the brain where it represents 15% of the total phospholipid pool. PS is used as a nutritional supplement for a long time. Historically it was obtained from the bovine cortex but due to potential infectious diseases, soy derived phosphatidylserine has become a safe alternative (Jorissen et al. 2002). Exogenous PS supplementation has shown benefits in physical exercise (Monteleone et al. 1990; Kingsley et al. 2006) and mental performance, where it is known for improving brain functions including long term memory and recognition especially in elderly people. Cenacchi et al. (1993) and Amaducci et al. (1988) both reported an improvement of cognitive performance in elderly people with cognitive decline respective Alzheimer disease after a PS-supplementation. Benton et al. (2001) demonstrated for the first time an effect of PS also on young adults. They showed that subjects which received PS supplementation were feeling less stressed and having a better mood during a cognitive task. In another study there is a discussion of using PS in ADHD children with benefits in attention and learning (Kidd 2000).

Therefore PS seemed to have a positive affect on cognitive functions including attention, concentration and recognition (Jäger et al. 2007), but nothing is known about the influence of PS after induced stress. Only Kingsley et al. (2006) investigated the influence of PS supplementation on oxidative stress during recovery following physical exercise/stress and found no significant effects.

To investigate that idea we use a double-blind placebo controlled paradigm where stress was induced before the subjects have to perform two cognitive tasks. In a Test-Retest design this was conducted before and after a chronic supplementation period (PS / placebo). To examine the underlying cortical mechanisms we measured EEG cortical activity before and after the induced stress. In association with attention and brain functions the EEG spectral

power analysis is a widely used method to evaluate changes in cortical activity in and after cognitive (Gevins et al. 1997, Klimesch et al. 1998; McEvoy et al. 2001) and sensorimotor tasks (Smith et al. 1999; Slobounov et al. 2000; Baumeister et al. 2007). In this context the Beta-1 spectral power plays a role in mental (Adey 1997; Diego et al. 2004) and physical stress (Mechau et al. 1998; Crabbe and Dishman 2004) whereas the frontal Theta and global Alpha-1 components are closely related to task-specific and unspecific attention (Gevins et al. 1997, Niedermeyer 2005).

It was the purpose of the current study to examine the effect of a chronic PS supplementation on i) cognitive performance and ii) cortical activity after induced stress. We hypothesized that a PS supplementation results in better cognitive performance and changes cortical activity compared to a control group.

## **Materials and Methods**

**Subjects.** A total of 16 male, right-handed volunteers ( $25\pm 3$  years;  $78.3\pm 6.9$  kg;  $184\pm 4$  cm) participated in the study completed all of the study requirements. The subjects were divided randomly in a PS ( $n=8$ ;  $24\pm 3$  years;  $77.0\pm 8.3$  kg;  $183\pm 4$  cm) and in a placebo group ( $n=8$ ;  $26\pm 2$  years;  $80.0\pm 5.6$  kg;  $185\pm 4$  cm). All subjects were recruited from the University of Paderborn. They all were non smokers and had no history of neurological, cardiovascular or other major disorders, no current use of medications or drugs and no physical or psychological exposures (e.g. intensive training sessions, examinations) 24 hours prior to the trials. All subjects had normal or corrected to normal vision at the time of the experiment. The participants were instructed to avoid changes in their every day behaviour (especially their diet). The study was done in accordance with the rules and regulations established by the Institutional Review Board for ethical treatment of human subjects. All subjects signed an informed consent after the explanation of the testing procedure. All data were treated with confidentiality.

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**Experimental design.** All subjects have had one preliminary visit to the laboratory to undergo a familiarization trial in order to avoid learning effects in the cognitive tasks before conducting the two main trials.

All participants then performed the two main trials, which were separated by exactly 42 days (6 weeks). The subjects were assigned, in a randomized, double-blind design, to receive either one nutritional bar (IQ PLUS brain bar, Giventis, Germany) per day containing 200 mg soy-based PS (PS group; n=8) or a corresponding placebo bar (control group; n=8). Each bar had a weight of 35g, providing 149 kcal, 4.8g protein, 20g carbohydrates, 5.5g fat and vitamins (1.4 mg vitamin B1, 1.4mg vitamin B6, 42mg vitamin C, 4.6mg vitamin E, 2.8mg niacin, 4.2mg pantothenic acid). The supplementation was started immediately after the first trial and was continued until the day before the second trial. During the supplementation period the subjects weighed and recorded their food three days a week to ensure the same diet over the supplementation phase.

**Main trial procedures.** On both test days (trial 1 and trial 2) all subjects received a standardized breakfast and were not allowed consuming caffeine or any other stimulants. On arriving in the lab the EEG electro cap and the HR belt (Polar, Sporttester, Germany) were attached to the subjects. They were asked to lie supine and relax for a 10 min period. After relaxing the participants were comfortably seated in a chair in front of a computer monitor with keyboard and mouse. The EEG baseline measurement (M1) was conducted at rest with eyes open for two minutes. To induce stress to the DAF (delayed Auditory Feedback) was performed. In this task the normal auditory feedback of speech is drowned and disturbed by a timed, delayed feedback (Novel SVG 3, NEG, Germany). The subject has to read sections from Immanuel Kant's "Kritik der reinen Vernunft" because of its complexity, so it can hardly be memorized and is therefore suitable for repeated use. They hear everything they have spoken after an adjustable delay. In this case the delay was set at 175 ms. Afterwards the cognitive tasks were performed in the described order stoop-colour word test and D2 test only interrupted by the EEG measurement (M2-M4; 2 min eyes open) immediately after each

task. Then there was a 10 min relaxing period and after that an EEG measurement with closed eyes (M5) was evaluated.

**Cognitive tasks.** The subjects had to perform two different cognitive tasks which include:

*Stroop colour-word interference test* (Irtel 1993). The stroop task was selected as a measure of inhibition, a key executive function. The task requires a suppression of response to a dominant stimulus pattern (printed words) while attending and responding to a secondary stimulus characteristic (ink colour). In the experimental session two words are presented on the monitor, the upper one of which is always written in a certain colour (red, blue, green, yellow), while the lower one is always in white. The test subject must decide whether the meaning of the lower word reflects the characteristic (ink colour) of the upper word. For a “correct” decision the subject has to push the left button of a computer mouse, for a “incorrect” decision the right button must be pressed. In the analysis three conditions appear: congruent congruent (meaning below and color above coincide), incongruent (meaning below does not coincide with the color above), and neutral (there is no connection between semantic category and the characteristic to be named – often meaningless sequences of letters). In total, 216 reactions times (72 in each condition) will be analyzed per test. The duration of the test was 20 minutes.

*D2 concentration test* (Brickenkamp 2002). The task was used to objectively document alertness, capacity to deal with stress and capacity of concentration. Within a preset time the subject must cross out specific letters that are arranged in rows: the lower case “d” with two dots above must be crossed out. Two types of mistakes can occur: omission (correct letters were not crossed out) and confusion (incorrect letters were crossed out). Total numbers of letters worked on was calculated as concentration performance (CP), omissions (O) and false answers (F) was described.

**Heart rate (HR) measures.** HR were continuously recorded at each 5-sec interval using short range telemetry (Polar Sporttester, Polar Electro, Finland). HR were averaged (M1-M5)

**Cortical (EEG) measures.** The EEG was recorded by a stretchable electro cap (ElectroCap Inc., USA) in accordance with standards of the international 10:20 System. At the baseline, directly after the induced stress, the cognitive performance of each task (Stroop, D2) and after 10 min recovery (M1-M5) EEG was recorded continuously for two minutes from 17 scalp locations (Fz, F3, F4, F7, F8, Cz, C3, C4, Pz, P3, P4, T3, T4, T5, T6, O1, O2) using the central electrode (Cz) as physical reference. All EEG data were recorded and stored using the CATEEM system (MediSyst, Linden, Germany). The signals were sampled at 512 Hz / 12 bit and amplified (DC=20 M $\Omega$ ). Before each measurement an impedance test ensured a sufficient signal to noise ratio. The physiological signals were highpass filtered at 0.86 Hz. An automatic artefact detection (depending on amplitude level and signal slope) was followed by a visual inspection. Only artefact free segments were used for analysis.

In the EEG signals Fast Fourier Transforms (FFT) were calculated on 50% overlapped, 512 sample Hanning windows at each electrode for all artefact free segments with the CATEEM system (MediSyst, Linden, Germany). The power spectra were divided into different frequencies: Theta (4.75 – 6.75 Hz), Alpha 1 (7.0 – 9.5 Hz), Alpha 2 (9.75-12.5 Hz) and Beta 1 (12.75 – 18.5 Hz). For the statistical analyses, a logarithmic (log) transformation of the power values was necessary to stabilize the variances (Pivik et al. 1993). Average log-transformed power spectra were computed across all time samples (M1 to M5) in both trials for each subject.

**Statistical Analysis.** For statistical analysis SPSS 12.0G software was used. All results are given as mean values and standard deviation. The Kolmogorov-Smirnov (K-S) test was used to determine if variables fit the Gaussian distribution. Subject characteristics were compared under supplementation groups using independent t-tests. To examine differences in peripheral and cortical parameters a mixed-model repeated-measures ANOVA (within-subject factors: trial and time; between-subjects factor: supplementation groups) was conducted. Compound symmetry, or sphericity, was verified by the Mauchley test. When the assumption of sphericity was not met, the significance of F-ratios was adjusted according to the Greenhouse-Geisser procedure. If a significant interaction was identified for group X trial

X time, a PS-supplementation effect was accepted. If a significant main effect of time appeared, multiple Bonferroni corrected paired t-tests were made. The outcome of statistical calculations were declared significant if  $p \leq 0.05$ . The partial  $\eta^2$  statistic provided estimates of the effect sizes in significant effects.

## RESULTS

**Cognitive task performance.** The results for cognitive testing are shown in table 1. During all tasks (stroop colour word test, D2 attention test) the subjects of both groups increase their performance significantly from trail 1 to trail 2 (trail effect, see tab.1). There were no significant between-groups differences (DAF (RW):  $F_{1,14}=0.527$ ,  $p=0.480$ ; stroop (neutral):  $F_{1,14}=0.464$ ,  $p=0.507$ , (congruent):  $F_{1,14}= 4.262$ ,  $p=0.058$ , (incongruent):  $F_{1,14}= 3.868$ ,  $p=0.078$ ; D2 (CP):  $F_{1,14}=0.823$ ,  $p=0.380$ , (F):  $F_{1,14}=0.590$ ,  $p=0.455$ , (O):  $F_{1,14}=0.256$ ,  $p=0.621$ ). There is no interaction effect of group x trail x time existent in any task parameter.

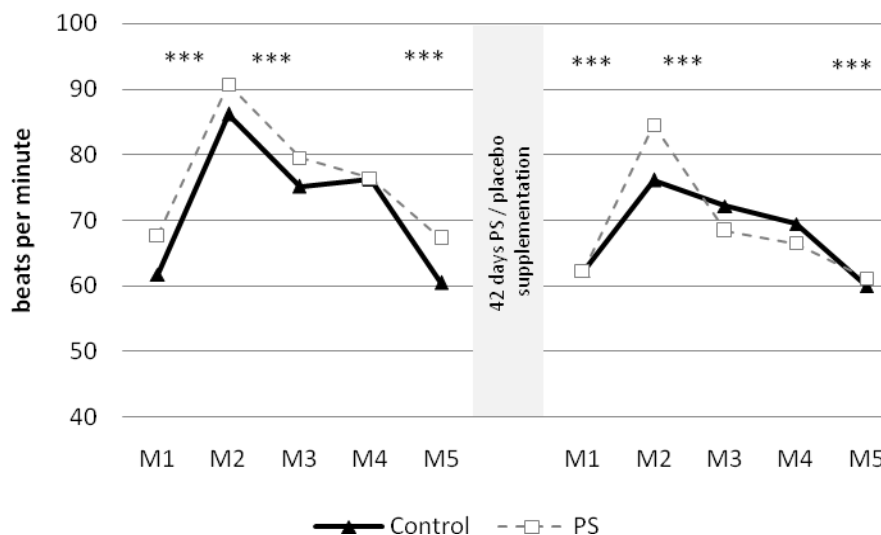


FIGURE 1. Heart rate (HR) of both groups during baseline (M1), induced stress (M2), Stroop (M3), D2 (M4) and after 10 min recovery (M5) before and after chronic supplementation period (post hoc significance level in both groups: \*  $p \leq 0.05$ , \*\*  $p \leq 0.01$ , \*\*\*  $p \leq 0.001$ )



**Heart rate measures.** The mean heart rate changes significantly within-subjects (within-factor time:  $F_{4,56} = 61.570$ ,  $p < 0.001$ , partial  $\eta^2 = 0.815$ , fig. 1). No significant between-subject effect is existent (between-factor group  $F_{1,14} = 0.280$ ,  $p = 0.605$ ) and PS-supplementation has no effect on HR values.

TABLE 1. Performance in cognitive tasks before and after chronic supplementation period

Test	Parameter	Trial	control	PS	main time effect		
			[ms]	[ms]	F	p	partial $\eta^2$
<b>Stroop</b>	congruent	1	807.8 ± 76.6	758.9 ± 77.9	13.327	.003	.488
		2	747.6 ± 58.2	680.5 ± 55.5			
	neutral	1	793.6 ± 63.7	771.7 ± 54.9	36.836	<	.725
		2	705.4 ± 54.2	691.0 ± 66.3			
	inkongruent	1	958.1 ± 64.8	850.6 ± 87.5	38.316	<	.732
		2	813.8 ± 84.2	762.1 ± 81.9			
<b>D2</b>	CP	1	620 ± 67	593 ± 41	27.852	<	.666
		2	667 ± 55	642 ± 68			
	O	1	6.88 ± 3.5	7.50 ± 4.1	10.978	.005	.440
		2	3.50 ± 2.2	4.75 ± 5.8			
	F	1	7.25 ± 3.7	8.63 ± 4.1	10.427	.006	.428
		2	3.63 ± 2.2	5.13 ± 6.2			

**Cortical activity (EEG) measures.** There was a main within-subject change over time (within-factor time) in different frequencies. In the Theta frequency significant power changes appear in frontal (Fz, F3, F4, F7, F8:  $F_{4,56} \geq 8.657$ ,  $p \leq 0.001$ , partial  $\eta^2 \geq 0.382$ ; fig. 2; post hoc Fz, F3, F4, F7 ( $p \leq .05$ ): M1-M5, M2-M5, M3-M5) and temporal (T3, T4, T5, T6:  $F_{4,56} \geq 4.365$ ,  $p \leq 0.004$ , partial  $\eta^2 \geq 0.238$ ; post hoc T3, T6 ( $p \leq .05$ ): M2-M5, M3-M5, M4-M5; post hoc T4 ( $p \leq .05$ ): M3-M5, M4-M5; post hoc T5 ( $p \leq .05$ ): M1-M5, M2-M5, M3-M5, M4-M5) regions. The Alpha-1 power values demonstrate significantly increased spectral power values over time in frontal (Fz, F3, F4, F7, F8:  $F_{4,56} \geq 9.319$ ,  $p < 0.001$ , partial  $\eta^2 \geq 0.400$ ; post hoc Fz, F3, F4, F7, F8 ( $p \leq .05$ ): M1-M5, M2-M5, M3-M5, M1-M4, M2-M4, M3-M4), central (Cz, C3, C4:  $F_{4,56} \geq 9.709$ ,  $p < 0.001$ , partial  $\eta^2 \geq 0.410$ ; post hoc Cz, C3, C4 ( $p \leq .05$ ): M1-M5, M2-M5, M3-M5, M1-M4, M2-M4), parietal (Pz, P3:  $F_{4,56} \geq 10.095$ ,  $p < 0.001$ , partial  $\eta^2 \geq 0.419$ ; post hoc Pz, P3 ( $p \leq .05$ ): M1-M5, M2-M5, M3-M5, M1-M4, M2-M4), and temporal (T3, T4, T5, T6:  $F_{4,56} \geq 9.364$ ,  $p < 0.001$ ,

partial  $\eta^2 \geq 0.400$ ; post hoc T3, T4, T5, T6 ( $p \leq 0.05$ ): M1-M4, M1-M5, M2-M5) electrode positions. Beta-1 power values increased over time at frontal (Fz:  $F_{4,56} = 12.310$ ,  $p < 0.001$ , partial  $\eta^2 = 0.468$ , post hoc ( $p \leq 0.05$ ): M1-M2, M1-M4, M1-M5, M2-M4; F3:  $F_{4,56} = 9.127$ ,  $p < 0.001$ , partial  $\eta^2 = 0.395$ , post hoc ( $p \leq 0.05$ ): M1-M4, M1-M5, M2-M5) and parietal (Pz:  $F_{4,56} = 12.157$ ,  $p < 0.001$ , partial  $\eta^2 = 0.465$ ; P3:  $F_{4,56} = 8.520$ ,  $p < 0.001$ , partial  $\eta^2 = 0.387$ , post hoc Pz, P3 ( $p \leq 0.05$ ): M1-M4, M1-M5, M2-M4, M2-M5, M3-M4, M3-M5) sites

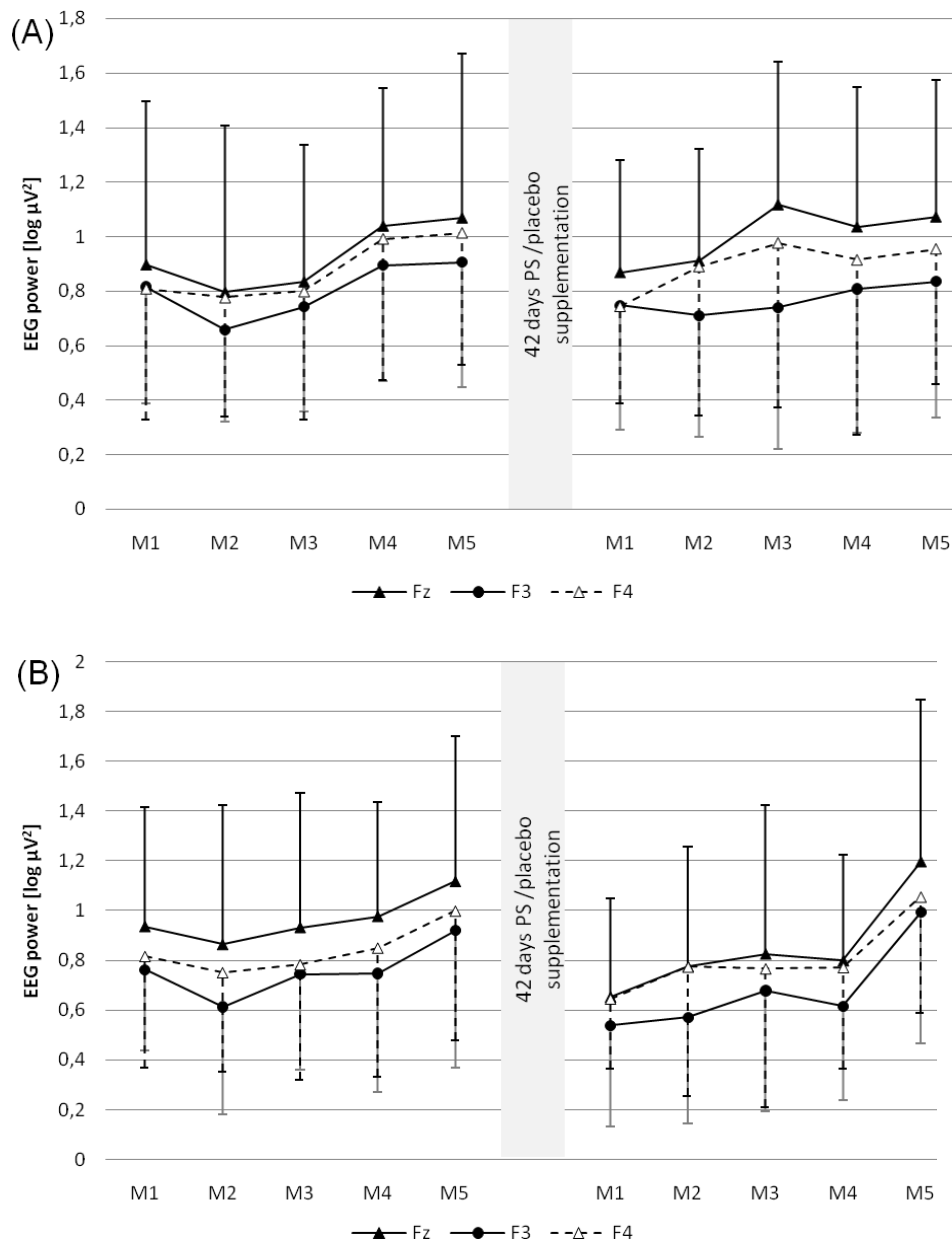


FIGURE 2. Overview of frontal (Fz, F3, F4) Theta power values of PS (A) and control (B) group immediately after rest (M1), induced stress (M2), Stroop (M3), D2 (M4) and 10 min recovery (M5) before and after chronic supplementation period (due to a better understanding we set aside description of significance in this diagram. See text for post hoc significance level)

Alpha-2 and Beta-2 show no significance in within-subject differences. Significant main group effects between the PS-group and the placebo group could not be detected in any frequency band.

The supplementation of PS has a significant effect (supplementation group x trial x time) in the Beta-1 right-frontal sites F4 ( $F_{4,56} = 3.324$ ,  $p = 0.016$ , partial  $\eta^2 = 0.192$ ; post hoc inner-subject factor trial ( $p \leq 0.05$ ): control M1 before - M1 after fig.3) and F8 ( $F_{4,56} = 3.101$ ,  $p = 0.022$ , partial  $\eta^2 = 0.181$ ; post hoc inner-subject factor trial ( $p \leq 0.05$ ): control M1 before - M1 after, between-subject factor group ( $p \leq 0.05$ ): M1 after, M3 after, fig.4)

## DISCUSSION

The main finding of the study was that supplementation of Phosphatidylserine administered for 42 days significantly decreases Beta-1 power in right hemispheric frontal brain regions before and after induced stress.

Increased beta-1 spectral power is described as an indicator of activation associated with cognitive tasks demands (Fernandez et al. 1995; Ray and Cole 1985), higher neurophysiological function (Thatcher et al. 1998). Adey (1997) described an increased frontal Beta-1 power in astronauts in association with an increased visual information overload, simulating hazardous flight conditions, as an "information overflow". Diego et al. (2004) investigated three types of relaxation (massage and vibrator effects) and have found a significant decrease in frontal Beta-1 activity. They interpreted the finding in connection with the participant's response to relaxation during the stimulation. Taken together these results it can be stated that a higher frontal neural activity is associated with an increased Beta-1 power and contrary a decrease in frontal Beta-1 power values demonstrated a form of relaxation (Niedermeyer 2005). In this study the PS supplementation lead to significant decreases in the right hemispheric frontal brain area before and after induced stress whereas the beta-1 power was higher in the control group indicating a higher activation state.

Therefore the PS group demonstrated a higher state of relaxation after and even before the induced stress which could not be detected before the supplementation period.

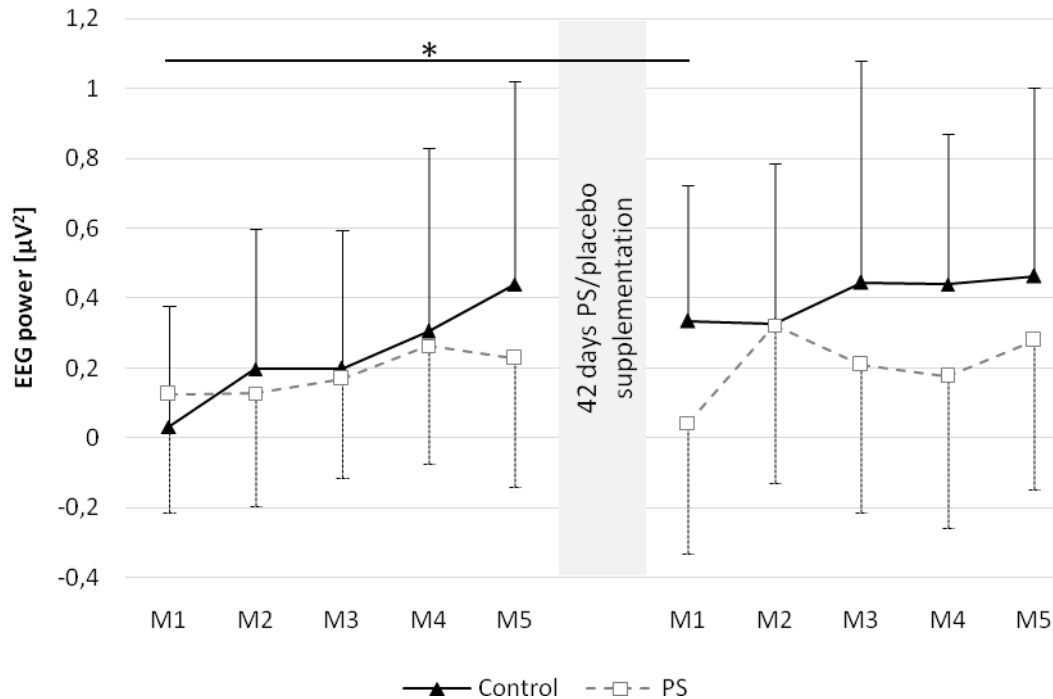


FIGURE 3. Right-frontal (F4) Beta-1 power values of both groups immediately after rest (M1), induced stress (M2), Stroop (M3) and D2 (M4) and 10 min recovery (M5) before and after chronic supplementation period (post hoc significance level: \*  $p \leq .05$ )

Frontal and prefrontal regions of the brain mediate executive processes (i.e. inhibition, selection, planning, attention, coordination, concentration; Sauseng et al. 2005; Bixby et al. 2007). Given the higher activation (expressed by higher Beta-1 activity) in frontal brain regions immediately after the cognitive tasks it can be speculated that more resources of executive functions were used in the control group due to the given mental stress by the delayed auditory feedback. It seemed that PS induced a more relaxed state which results in a suppressed frontal Beta-1 activity. In conjunction with an “information overflow” (Adey 1997) it seemed that the controls without any supplementation are not able to relax and probably still engaged in processing information still focusing on the performed task and activating frontal resources for executive functions.

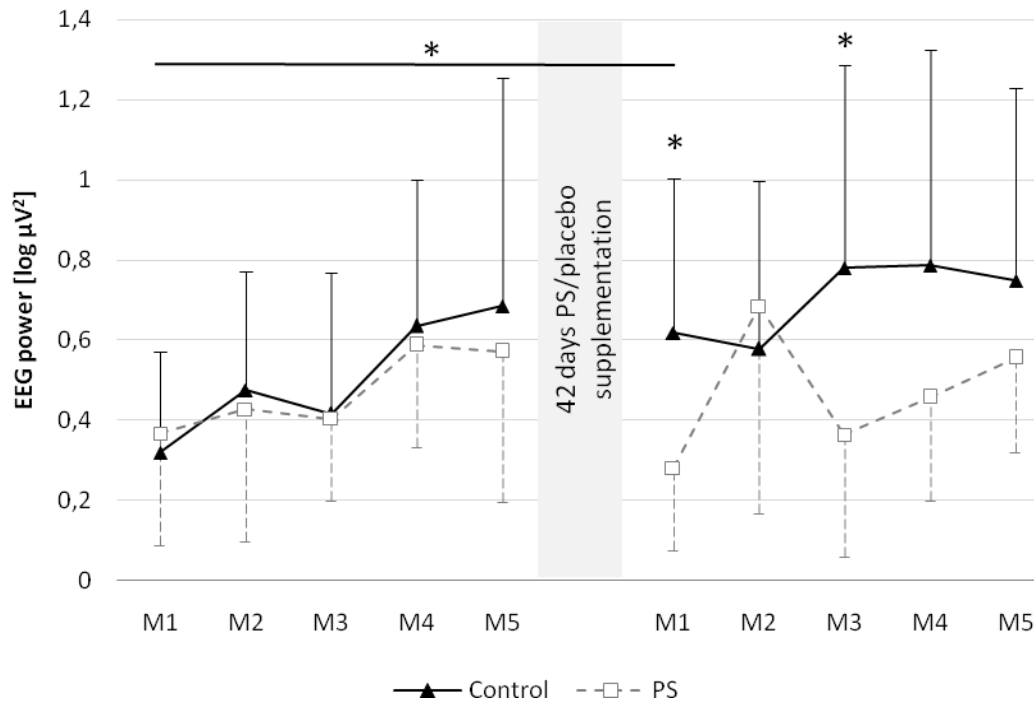


FIGURE 4. Right-frontal (F8) Beta-1 power values of both groups immediately after rest (M1), induced stress (M2), Stroop (M3) and D2 (M4) and 10 min recovery (M5) before and after chronic supplementation period (post hoc significance level: \*  $p \leq 0.05$ )

The cognitive performance in the Stroop colour-word test and D2-test improved after the supplementation period in the PS group as well as in the control subjects. This may be due to a familiarization effect. The participants know the situation and tasks during the second trial and therefore may have reached better results. The supplementation of PS has a long history in age-related diseases to improve cognitive performance in memory and learning. Crook et al. (1991) divided patients with age-related memory impairment in an PS supplementation group and controls (placebo). After a 12 week intervention the PS group performed better in memory and learning tasks relative to the controls. Although clinical trials demonstrated some improvements in memory performance in Alzheimer's patients (Amaducci et al. 1988) only Benton et al. (2001) proofed an effect of PS in young healthy subjects during arithmetic tasks. There were no differences in task performance but the PS-supplemented participants reached the same results and feeling less stressed compared to a

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control group. To conclude the results in this study concerning the effect of Phosphatidylserine, the PS group is able to perform as good as the control group in a more relaxed state indicated by the frontal Beta-1 spectral power values. To elucidate the described effect more research is needed to investigate the underlying mechanism.

Both groups were able to perform better in all given tasks after the supplementation period. Given the highest activation immediately after the delayed auditory feedback in both groups one can assume that stress was really induced. This is supported by the significant increases before and decreases after the DAF in heart rate (HR) during the tasks.

Independent from the supplementation of PS the study regime demonstrated significant increases after the induced stress in different frequency bands (Theta and Alpha-1). The frontal Theta power tended to result in increased values after the DAF over time in both groups. Recent data indicated a generation of frontal Theta in the anterior cingulate cortex (Gevins et al. 1997). Neuroimaging and brain lesion studies have shown that this anatomical region is an important component of the human attentional system (Posner and Petersen 1990; McEvoy et al. 2001). Different authors have found that frontal Theta increases when task performance and attention must be sustained over time (Nakashima and Sato 1992; Gevins et al. 1997; Laukka et al. 1995; Smith et al. 1999). Therefore, the attentional resources at the end (M5) of the test seemed to be increased compared to the beginning independently from PS-supplementation. Furthermore, the frontal Theta power increases after the stress task indicating a higher attention level.

In addition, the results show an increase over time in the slow Alpha-1 frequency band at all electrode positions in all regions of the brain. Alpha is the dominant frequency in the human scalp of EEG of adults. Alpha activity is often described as a form of cortical idling with its amplitude inversely related to the number of neuronal populations activated during cognitive and motor processes (Hatfield et al. 2004; Niedermeyer 2005). The slow alpha band component (Alpha-1) is attenuated over broad regions of the cortex and is a nonspecific attentional and expectancy processes parameter. An increase in the Alpha-1 frequency band

over time supports the idea of a decrease of non-specific attention over time but PS-supplementation plays no role in this during the trials.

## **CONCLUSION**

It can be concluded that a 42 days chronic PS supplementation influenced the frontal Beta-1 frequency before and after induced stress. Both groups were able to perform better in cognitive tasks after the supplementation period with a decreased activation in frontal brain regions immediately after the tasks in the PS group compared to the controls. To our knowledge this is the first study to show these effects of PS indicating a more relaxed state after induced stress. Further research is required to confirm this findings.

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## CHAPTER 7

### **Sensorimotor Control and Brain Activity in sports-related conditions**

Understanding the physiological process of expertise has been a key challenge in sport performances for decades in sports medicine research. Once the neural mechanism behind superior motor behavior is understood we may be able to better clarify and evaluate the training process in terms of a holistic approach including different biological systems (e.g. cardiovascular, musculoskeletal and nervous system). Different environments may also play an important role in this picture concerning comparable results in real- world environments versus laboratory settings.

Three publications in this research program demonstrate that sports-related conditions cause variations in EEG power due to the working-memory-hypothesis in sensorimotor control in an expertise approach regarding skill levels and according to different environments.

Experts and novices were investigated in a standardized golf putting task (Publication 4). During a 5x4 min putting series EEG data was collected from 20 participants and EEG spectral power was calculated. The expert golfers achieved significantly better scores compared to the novices. Brain activity demonstrates a sensitivity of EEG power. Putting expertise seems to be associated with significant higher Theta values in frontal brain areas and higher parietal Alpha-2 power compared to the novices. According to the working-memory-hypothesis the findings suggest that expert golfers have developed action strategies including attentional control and a neural efficiency in parietal sensory information processing.

In a further investigation the effect of different environments on brain activity during putting performance was investigated by comparing a real-world environment to a virtual reality condition (Publication 5). The participants performed significantly better in the real-world environment compared to the virtual reality which uses the Nintendo Wii. Differences in EEG

power during putting between the environments fit the working-memory-hypothesis. The participants performed with higher frontal Theta power values and increased parietal Alpha-2 power in the real-world environment. The findings suggest that putting performance and brain activity related to the working memory was influenced by the choice of environment. After the demonstration of the feasibility of EEG measures during golf putting in a laboratory based environment, this methodological study investigates the differences between a laboratory setting and a real-world environment of playing golf, namely the golf course (Publication 6). Eleven participants performed standardized golf puttings. EEG was measured during the performance and compared between both environments. Frontal Theta (F4) was the only electrode position which demonstrates significantly higher values in the real-world environment. Therefore we assume that results in EEG power obtained during putting in the laboratory can be transferred to the real-world environment.

The golf putt seems to be a good model for a sports-related complex task, because it is feasible to obtain reliable EEG data especially in the real world environment. Sports related conditions affect the brain state related to the working-memory-hypothesis. Furthermore, a longitudinal study is needed to verify the results in light of the training process to get a deeper insight into how training can be optimized and evaluated in terms of quality and intensity from a neuronal perspective.

## PUBLICATION 4

Baumeister, J., Reinecke, K., Liesen, H., & Weiss, M. (2008). Cortical activity of skilled performance in a complex sports related motor task.

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<b><i>Acquisition of data:</i></b>	<b>Baumeister, Reinecke</b>
<b><i>Analysis and interpretation</i></b>	<b>Baumeister</b>
<b><i>Drafting the manuscript</i></b>	<b>Baumeister</b>
<b><i>Critical intellectual revision</i></b>	<b>Reinecke, Liesen, Weiss</b>
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## ABSTRACT

A skilled player in goal-directed sports performance has the ability to process internal and external information in an effective manner and decide which pieces of information are important and which are irrelevant. Focused attention and somatosensory information processing play a crucial role in this process. Electroencephalographic (EEG) recordings are able to demonstrate cortical changes in conjunction with this concept and were examined during a golf putting performance in an expert-novice paradigm. The success in putting (score) and performance-related cortical activity were recorded with an EEG during a 5 x 4 min putting series. Subjects were asked to putt balls for four min at their own pace. The EEG data was divided into different frequencies: Theta (4.75 – 6.75 Hz), Alpha-1 (7 – 9.5 Hz), Alpha-2 (9.75-12.5 Hz) and Beta-1 (12.75 – 18.5 Hz) and performance related power values were calculated. Statistical analysis shows significant better performance in the expert golfers ( $p < .001$ ). This was associated with higher fronto-midline Theta power ( $p < .05$ ) and higher parietal Alpha-2 power values ( $p < .05$ ) compared to the novices in golf putting. Frontal Theta and parietal Alpha-2 spectral power in the ongoing EEG demonstrate differences due to skill level. Furthermore the findings suggest that with increasing skill level, golfers have developed task solving strategies including focussed attention and an economy in parietal sensory information processing which lead to more successful performance. In a theoretical framework both cortical parameters may play a role in the concept of the working memory.

**Key words:** EEG, skill level, working memory, attention, information processing, golf

## INTRODUCTION

Understanding the process of skill acquisition has been a key challenge in sports performances for decades. Once the process behind motor behaviour is understood intervention programs may be clarified to optimize skill acquisition. A direct approach in sports performance to examine differences in skill levels is to compare beginners and experts in a specific task. The novice state is characterized by stimulus identification which involves mostly perceptual processes to find the relevant internal and external information for the motor task whereas experts already can filter information and focus on decision and strategy processes (Wrisberg 2001). This processing of different information involves the handling of external and internal information as well as attentional resources which are fundamental in this process (Abernathy 2001). It takes place at the brain level where measuring in a complex sports related motor task is always difficult.

Nevertheless the electroencephalography (EEG) has been shown to be able to monitor electro-cortical changes in these situations. The most used approach in the field of sports performance have drawn attention to the important conjunction between performance and the preparatory period in archery (Salazar et al. 1990), rifle shooting (Hatfield et al. 1984, Häufner et al. 2000) and golf (Crews & Landers 1993).

Beside that the EEG is well suited to demonstrate differences in brain-state which have been described when performers adopt an effective strategy and develop a skill in cognitive, visuomotor, sensorimotor tasks (Smith et al. 1999, Slobounov et al. 2001, Grunwald et al. 2001, Baumeister et al. 2007).

The putting performance in golf is a complex, goal-directed sports performance and offers the possibility to get reliable data as have been shown before by other investigators (Crews & Landers 1993; Babiloni et al. 2008). Therefore our approach focussed on the oscillatory activity during golf putting performance over a longer period: the absolute performance-related power (PRPow). Changes in the brain state during continuous tasks as a whole were investigated rather than phasic changes referred to as 'event-related'.

In more detail different authors had described the frontal Theta power values as an indicator of attention and the parietal Alpha-2 spectral power related to somatosensory information processing (Gevins et al. 1997; Slobounov et al. 2000; Grunwald et al. 2002; Baumeister et al. 2007). Smith et al. (1999) reported increased frontal Theta power associated with task complexity and focussed attention and decreased parietal Alpha-2 values with increased information processing in a cognitive (3-back working memory task) and visuomotor task (video game 'space fortress'). In a sensorimotor task where ACL patients had to reproduce knee angles our research group (Baumeister et al. 2007) had shown that ACL patients have higher frontal Theta power and lower parietal Alpha-2 power as their healthy counterparts. We interpreted the results with higher focussed attention and higher activity of neural populations in the somatosensory cortex associated with an increased information processing.

Therefore it remains unclear if this could be shown during a complex, goal-directed sports performance. The putting performance in golf is well suited to serve as a complex, goal-directed sports performance and offers the possibility to get reliable data as have been shown before by other investigators (Crews & Landers 1993; Babiloni et al. 2008).

In this context the aim of this study is to examine differences related to EEG spectral power depending on the skill level during a golf putting performance. Therefore we assume that experts and novices in golf putting differ in frontal Theta and parietal Alpha-2 power values.

## **METHODS**

### **Subjects**

Nine male, right handed experienced golfers ( $26.4 \pm 4.1$  years;  $86.7 \pm 4.8$  kg;  $189.4 \pm 2.1$  cm;  $7.6 \pm 4.2$  years golf experience; playing golf  $13.0 \pm 7.0$  hours/week) with golf handicap of  $8.3 \pm 7.5$  were compared to nine male, right handed novice volunteers ( $24.6 \pm 3.4$  years;  $82.6 \pm 3.1$  kg;  $184.3 \pm 7.1$  cm). None of the novices had any experience with golf and putting. All participants were screened with a health questionnaire and had no history of neurological,

cardiovascular or other major disorders, no current use of medications or drugs and no physical or psychological exposures (e.g. intensive training sessions, examinations) 24 hours prior to the trial. All subjects had normal or corrected to normal vision at the time of the experiment. The study was done in accordance with the rules and regulations established by the Institutional Review Board for ethical treatment of human subjects. All subjects signed an informed consent after the explanation of the testing procedure. All data were treated with confidentiality.

### *Procedures*

The study was conducted at a laboratory at the Institute of Sports Medicine (University of Paderborn). In a preliminary examination the participants were familiarized with the surroundings, the task and procedures. After measuring an EEG at rest subjects were instructed to putt 10 golf balls (Bridgestone, USA) across the carpet without any target to avoid a learning effect. Afterwards they completed the health questionnaire.

One week later the main part of the study was performed. On arriving at the laboratory the electro cap (ElectroCap Inc., USA) was attached to the subjects head. They were asked to lie supine and relax for a 10 minute period. Directly after relaxing, EEG signals were recorded for 2 min (M0). Then participants were required to putt golf balls (Bridgestone, USA) across a carpet towards a target (3m distance) for 4 minutes. They were instructed to do this as accurate as possible, at their own pace. No practice putts were allowed. During the four minute putting performance the EEG signals were recorded. This was repeated five times (M1-M5). Each performance period was followed by a rest in standardized sitting position for 2 min.

### *Measures*

**Performance.** All participants had to putt golf balls with a standardized Putter (UG-LE, Ping, USA) as accurately as possible from a 3m distance on a green carpet (ENIA Carpet,



Germany; stimp meter rate: 9 – mean comparable speed to medium fast greens) into a target which represented real hole size. Around the hole there were 9 x 9 squares (each 4,5 x 4,5 inch) marked with a different score depending on the proximity to the target hole with score=0 (lowest) and score=5 (highest). The sum of all putt-scores during each putting performance was divided by the number of putts. There was no verbal feedback given to the participants but they had their visual feedback after each stroke.

**Psychometric measures.** State and trait anxiety were measured using the Spielberger State–Trait Anxiety Inventory (STAI; Laux et al. 1980). The STAI is a reliable and valid measure that has been used with both clinical and nonclinical populations (Laux et al., 1980). The measure comprises separate self-report scales for assessing state and trait anxiety. The state anxiety scale consists of 20 items that evaluate current feelings of tension, anxiety, and nervousness, while the 20-item trait scale assesses anxiety levels in general.

**Subjective stress level.** The subjective stress level was recorded with the help of a visual analogue scale (VAS) (McCormack et al. 1988; Petru et al. 2005). The subjects were asked to estimate their state of stress on a scale-line with two end points from “no stress at all” (0 points) and “highest stress level” (10 points)

**EEG preparation and recordings.** The EEG was recorded by a stretchable electro cap (ElectroCap Inc., USA) in accordance with standards of the INTERNATIONAL 10:20 system (Jasper 1958). During the task performance (4 min) EEG was recorded continuously from 13 scalp locations (Fz, F3, F4, Cz, C3, C4, Pz, P3, P4, T3, T4, T5, T6) using Cz as a common average reference (Lehmann 1987) and a midforehead placement of the ground electrode (Pivik et al. 1993). All EEG data were recorded and stored using the CATEEM system (MediSyst, Linden, Germany). The signals were sampled at 512 Hz / 12 bit and amplified (DC=20 M $\Omega$ ). Before each measurement an impedance test ensured a sufficient signal to noise ratio (Schober et al. 1995). The physiological signals were high pass filtered at 0.86 Hz. Due to the problem of eye-head movements automatic artefact detection (depending on artefact rejection limit with respect to the EEG signal amplitude and slew rate limit with respect to the slew rate of the EEG signal; Schober et al. 1995) was completed followed by a

visual inspection by experienced electroencephalographers. Only artefact-free segments were used for analysis. In the EEG signals, Fast Fourier Transforms (FFT) were calculated on 50% overlapped, 512 sample Hanning windows for all artefact free segments with the CATEEM system (MediSyst, Linden, Germany). Performance-related power (PRPow) was defined as the mean absolute power during each task performance and was divided into different frequencies: Theta (4.75 – 6.75 Hz), Alpha 1 (7 – 9.5 Hz), Alpha 2 (9.75-12.5 Hz) and Beta 1 (12.75 – 18.5 Hz) Beta 2 (18.75 – 35 Hz). For the statistical analysis, a logarithmic (log) transformation of the power values was used to stabilize variances across subjects (Pivik et al. 1993). The transformed data in the different frequency bands were computed across all trials for performance (M1-M5) in each subject.

#### *Statistical Analysis.*

For statistical analysis SPSS 14.0G software was used. All results are given as mean values and standard deviation. The Kolmogorov-Smirnov (K-S) test was used to determine if variables fit the Gaussian distribution. Subject characteristics were compared under skill level groups using independent t-tests. To examine changes in psychometric / subjective stress measures and PRPow in the different frequencies an ANOVA with repeated measures (between-subject factor: group x within-subject factor: time) was conducted. Compound symmetry, or sphericity, was verified by the Mauchly test. When the assumption of sphericity was not met, the significance of F-ratios was adjusted according to the Greenhouse-Geisser procedure. Independent t-tests were used post hoc to detect significance between groups. If a significant main effect of time appeared, paired t-tests were made. The outcome of statistical calculations were declared significant if  $p \leq 0.05$ . A Bonferroni adjustment was applied to planned comparisons to minimize the likelihood of a type I error. The partial  $\eta^2$  statistic provided estimates of the effect sizes.

## RESULTS

**Performance.** The experts achieved significantly better scores in all five performances of a golf putting task compared to the novices (factor group:  $F_{1,16}=114.328$ ;  $p<.001$ ; effect size=.864; fig.1). The putting score changes significantly within-subjects (factor time:  $F_{4,72}=11.961$ ,  $p<.001$ , effect size =.399; post hoc M1 vs. M2/M3/M4/M5  $p\leq.001$ ). The average number of putts was  $18.7\pm 0.76$  in the novice and  $19.53\pm 1.05$  in the expert group in each 4 min performance period.

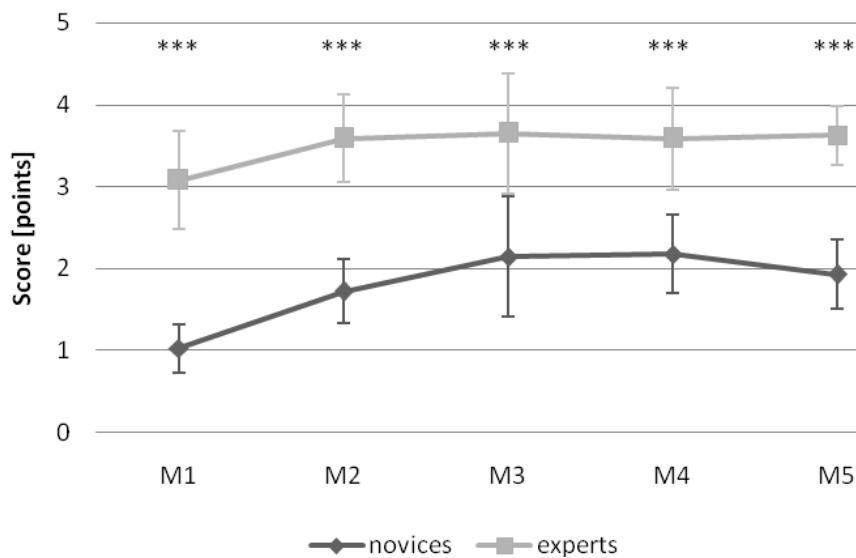


FIGURE 1. Score of novice and expert golfers during five putting performances

**Psychometric Measures.** The State-Trait Anxiety scores showed no differences between- (factor group) and within-groups (factor time) neither for the state (X1) nor for the trait (X2) parameters (tab.1).

**Subjective stress measures.** The subjective stress level showed no differences between- groups and within-subject main effect (factor time:  $F_{4,72}= 2.294$ ,  $p=.028$ , effect size =.155; post hoc: n.s.) with subjective stress values increasing over time in both groups (tab.1).

**EEG Parameters.** A comparison of all EEG parameters in the resting state (M0) between both groups showed no significant differences. EEG PRPow shows differences between

experts and novices in the Theta, the slow (Alpha-1) and fast Alpha (Alpha-2) frequency band. Task related Theta power shows significant higher values in the expert group compared to the novices at frontal Fz ( $F_{1,16}=5.275$ ;  $p=.035$ ; effect size  $=.248$ ; Fig. 2) and parietal Pz ( $F_{1,16}=7.856$ ;  $p=.013$ ; effect size $=.329$ ; post hoc M1:  $p=.031$ , M2:  $p=.005$ , M3:  $p=.021$ , M4:  $p=.001$ , M5:  $p=.021$ ) brain areas.

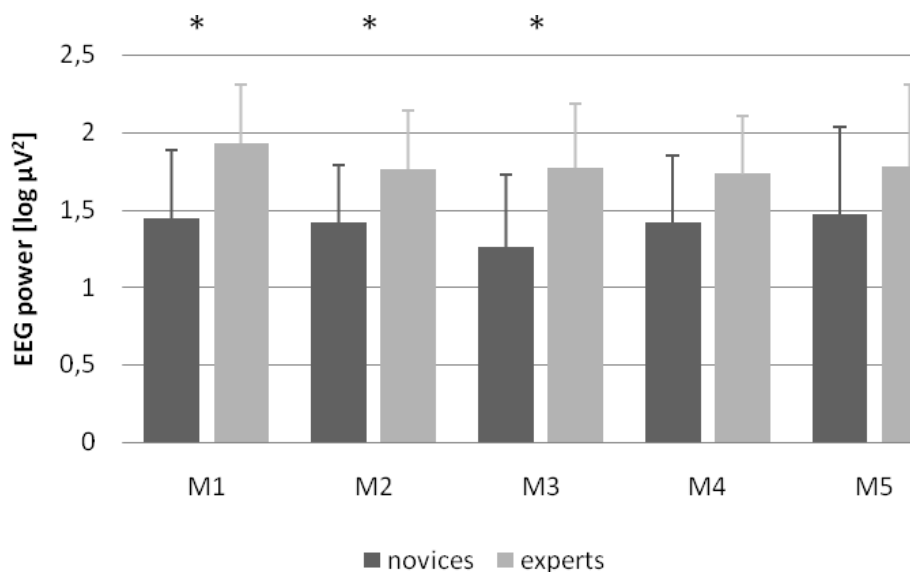


FIGURE 2. Frontal-midline (Fz) Theta power during putting performance (M1-M5) in experts and novices

(level of significance \*  $p<0.05$ )

Additionally the experts demonstrate significantly higher power in the Alpha-1 frequency band at the parieto-midline area (Pz,  $F_{1,16}=7.690$ ;  $p=.014$ ; effect size  $=.325$ ; post hoc M1:  $p=.039$ , M2:  $p=.016$ , M3:  $p=.033$ , M4:  $p=.006$ ).

Further analysis revealed that the experts exhibit significantly more PRPow at parieto-midline in the Alpha-2 frequency band (Pz) than novices ( $F_{1,16}=8.482$ ;  $p=.010$ ; effect size  $=.346$ ; Fig. 3). There were no group effects in the Beta-1 PRPow values and furthermore no within-subjects (factor time) or interaction effects (group x time) in any analyzed frequency.

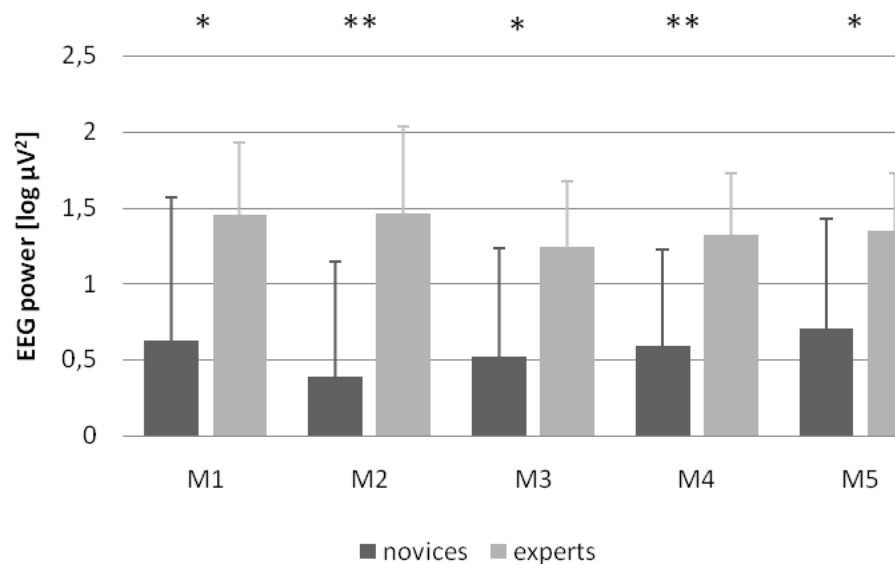


FIGURE 3. Parietal-midline (Pz) Alpha-2 power during putting performance (M1-M5) in experts and novices

(level of significance \*  $p < 0.05$ ; \*\*  $p < 0.01$ )

## DISCUSSION

The comparison of central activation in novice and expert golfers during putting performance was associated with significant changes in prominent spectral features in the ongoing EEG: frontal Theta and parietal Alpha-2 power. The performance data indicate that experts perform the putting task with a significantly better score compared to the novices. The performance score and the higher subjective stress level over time measured by a visual analogue scale suggest that both groups were engaged in the task and try to perform at their best level.

### *Theta PRPow*

The expert golfer group demonstrated higher fronto-midline (fm) Theta PRPow compared to the novices. This finding was consistent with numerous studies indicating a role of this frequency associated with attention in cognitive (Smith et al. 1999), visuomotor (Slobounov et al. 2001; Grunwald et al. 2002) and sensorimotor tasks (Baumeister et al. 2007). Results from other studies demonstrated a generation of fm Theta in the anterior cingulate cortex (Gevins et al. 1997). Neuroimaging and brain lesion studies have shown that this anatomical

region is an important component of the human attentional system (Karni et al. 1998, Luks et al. 2002). It has been suggested that fm Theta is related to focussed attention (Smith et al. 1999, Häufler et al. 2000). In the current study the experts performed with increased frontal-midline Theta power compared to the novices which might reflect a higher focussed attention during the putting performance. Speculations could lead to the idea that a higher level of concentration is needed for the experts to select the relevant information for a good performance in permanently changing situations which is a principle of the golf game. This focused attention is crucial for central executive functions like decision making and strategy selection. In contrast to that it could be speculated that novices might have no idea what information were relevant. Hence, there was no increased level of focused attention in golf putting beginners. The results were supported by Häufler et al. (2000) who found higher Theta power values in marksmen compared to beginners during a shooting exhibition. They concluded that expert exhibition is characterized by a state of focussed concentration.

There is only little reference on parietal Theta PRPow differences (Pz). Fournier et al. (1999) found increased Theta power in posterior brain areas with increased task complexity during a complex visuomotor task. The authors suggest that parietal Theta activity was higher with higher cognitive and behavioral demands. This is supported by Dolce et al. (1974) who found higher parieto-occipital Theta power in a reading task compared to an easy mental task. However the data suggests that higher parietal Theta power seems to be associated to higher skill level during a complex sports performance like the golf putt.

### *Alpha PRPow*

The second prominent frequency differentiating the skill level in this novice-expert paradigm was the Alpha frequency. Alpha is the dominant frequency in the human scalp in the EEG of adults and is thought to be generated in widespread areas of the cortex through thalamo-cortical interactions. Alpha activity is often described as a form of cortical idling with its amplitude inversely related to the number of neuronal populations activated during cognitive

and motor processes (Niedermeyer & da Silva 2005). Usually the Alpha frequency is divided into two subgroups: a “slow” (Alpha-1, 7.0-9.5 Hz) and a “fast” (Alpha-2, 9.75-12.5 Hz) component. The slow alpha band component (Alpha-1) is attenuated over broad regions of the cortex and is thought to be a nonspecific attentional and expectancy processes indicator. Lower values in the frontal Alpha-1 task related spectral power may reflect a global activation and therefore a higher unspecific arousal level in the novice group. It may indicate that the novices try to get a higher arousal on a global level. It can be speculated that this higher arousal level may describe the first step in skill acquisition. The novices in golf putting are activated but have no knowledge about the relevant information for their putting performance. Therefore they were not focused on special information. This was supported by the results of the frontal Theta PRPow where novices have lower values and were described as not focused compared to the expert group.

The fast Alpha (Alpha-2) reflects task-specific demands in parieto-occipetal regions in the area of the somatosensory cortex (Gevins et al. 1997, Smith et al. 1999). The higher PRPow in the expert group indicates the notion of less cortical activation in the parietal regions compared to the novices. That means the novices must actively process unfamiliar cues that seem to require more neuronal resources in the parietal cortex compared to the expert golfers. These findings are consistent with the results of Gevins et al. (1997) who described practice-related increases in Alpha-2 power with fewer cortical resources involved after skill development in a cognitive task. Similar results are described for visuomotor tasks. Smith et al. (1999) show enhanced Alpha-2 power values in conjunction with acquisition of a video tracking skill. They suggested that the development of task specific neurocognitive strategies might be reflected in regional differences in the alpha rhythm. In accordance with this Baumeister et al. (2007) found decreased Alpha-2 power in anterior cruciate ligament (ACL) patients in a somatosensory knee angle reproduction task at parietal regions of the brain representing the somatosensory cortex. They described their findings with a higher somatosensory information processing due to the injury in the ACL patients.

To put the results in a bigger frame the brain provides a tool which is responsible for information processing, focused attention and decision making: the working memory. The concept of the working memory suggests that a limited capacity system temporarily maintains and stores information, supports human thought processes by providing an interface between perception, long-term memory and action (Baddeley 2003). In this theoretical framework the central executive (CE) is assumed to be an attentional control system responsible for strategy selection, control and coordination of motor tasks with crucial importance for planning and decision making. There are already some references which connect the findings in frontal Theta power and parietal Alpha-2 power to the load of the working memory and in more detail to focused attention and information processing. In a cognitive task Sauseng et al. (2005) demonstrated that frontal Theta and parietal Alpha-2 power act in a fronto-parietal network which represents central executive functions in the concept of working memory. This study may be an approach to link the cortical activation of complex sports related motor performance and the idea of working memory. Further investigations should be conducted in this field to verify this idea.

## **SUMMARY**

In summary, the results of this study show that absolute performance related power (PRPow) values measured by EEG were associated with skill level in a golf putting task. The frontal Theta and parietal Alpha-2 spectral power seemed to be sensitive to this task. Furthermore the findings suggest that with increasing skill level, golfers have developed task solving strategies which came along with focussed attention and an economy in neural activity during sensory information processing in the somatosensory cortex. With the theoretical framework of the working memory it seemed to be possible to develop a model for goal directed sports performance like golf, rifle shooting or archery. Thus, in follow up studies controlled sensorimotor experiments (e.g. according to "isolated" senses like joint position



sense tasks or visual tasks) should be designed to control and manipulate the working memory load to enhance, accept or reject this model.

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## PUBLICATION 5

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<b><i>Analysis and interpretation</i></b>	<b>Baumeister, Weiss</b>
<b><i>Drafting the manuscript</i></b>	<b>Baumeister</b>
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## ABSTRACT

Low budget virtual environments like the Nintendo Wii increased in popularity and may play a role in motor learning related to sports and exercise. But nothing was known about the comparability of cortical activity of motor tasks in real and virtual environments. The aim of the study was to examine cortical differences between real and Wii based virtual sports performances using the golf putt as a model.

Ten male golfers (26.0±0.7 years; 81.8±5.6 kg; 184.5±6.0 cm; handicap 30.0±10.0; 2.9±1.0 years of golf experience) were asked to putt for 3min in random order in the real and the virtual Wii condition. A rest in sitting position (3min) followed each performance. The score and cortical activity (EEG) were recorded continuously.

The participants performed with a significant better score in the real condition ( $p \leq 0.01$ ). Compared to virtual putting Theta spectral power show a significant increase during real performance at F3 and F4 ( $p \leq 0.05$ ). Significantly increased Alpha-2 power was demonstrated during real putting compared to the virtual putting performance at P3 ( $p \leq 0.05$ ). The findings suggested that putting performance and brain activity was influenced by the choice of a real or virtual environment. The results were discussed based on the concept of the working memory where increased frontal Theta power indicated higher focused attention and higher Alpha-2 power was inversely related to the quantity of sensory information processing in the real putting compared to the virtual condition.

**Key words:** Golf, computer games, EEG, working memory

## INTRODUCTION

In the last decade the field of learning in a virtual environment had grown immensely. Practical applications for the use of this technology encompassed many fields from industrial training in machine operations to medicine, where surgeons could be trained in surgical techniques.

A virtual environment is a simulation of a real world environment that is generated through computer software and experienced by the user through a human-machine interface. An example for a low budget and simple to use virtual environment is the Nintendo Wii where the player used a remote control with motion sensors as a human-machine interface which transferred movements in the virtual environment. While energy expenditure had been a research focus in different situations while playing Nintendo Wii sports related games [10, 11] nothing is known about cortical activity associated with motor control.

Motor control of complex behaviours provided a common ground for researchers working on motor skill learning. Sensory feedback was a key factor in motor control in the real world as well as in virtual environments. In the central nervous system (CNS) feedback about performances is provided by different internal and external senses (e.g. vision, proprioception) and their processing in different areas of the brain. The working memory (WM) played a crucial role in this context. The concept of the working memory suggest that a limited capacity system temporarily maintains and stores information and supported human thought processes by providing an interface between perception, long-term memory and action [2]. In this theoretical framework the central executive (CE) is assumed to be an attentional control system responsible for strategy selection, control and coordination of motor tasks with crucial importance for planning and decision making. Human neuroimaging studies indicated that motor tasks requiring working memory activated a functional network linking frontal and parietal regions of the brain [21]. The activation of this network could be characterized by neuroelectric activity recorded by electroencephalographic (EEG) measurements. Changes in WM related information processing in cognitive, sensorimotor and complex motor tasks produced characteristic changes in amplitude of spectral

components of the ongoing EEG. In this context frontal Theta frequency was described as enhanced in tasks with greater central executive control demands with increased power values in tasks that required sustained focussed attention [25, 4, 5, 6, 8]. Conversely, the “fast” Alpha frequency (alpha-2: 9.75-12.5 Hz) in the parietal cortex seemed to be sensitive to the amount of somatosensory information processing with spectral power values inversely related to the activation of neuronal populations in this brain area [18]. Both frontal Theta and parietal Alpha-2 tended to increase as subjects develop skills in task performance [9, 17]. To support this Baumeister et al. [5] had been able to demonstrate differences according to EEG spectral power in an expert-novice paradigm during a complex sports-related task using the golf putt as a model. Experts demonstrated a higher score and have shown increased frontal Theta and increased parietal Alpha-2 power compared to the golf beginners. Other authors had also demonstrated the golf putt as a reliable model to measure cortical activity during the task [7, 1]. To match the real to a virtual condition Nintendo Wii provided a Golf putting game which could be standardized to real putting conditions in slopes and distance. From a theoretical point of view the perception and therefore sensory information processing should have changed while putting in the virtual environment. The perception of body position in space was the result of integration and interpretation of multiple sensory variables (especially vision). The angle under which the object was observed plays an essential role. Altering the angle can affect the participant’s estimation of distances [3] and forces [26] which were based on the proprioceptive modalities and processed in the working memory. In this case the EEG would be able to detect these differences.

Despite the intriguing EEG changes observed in the golf putting task between experts and novices, the oscillatory brain activation in a virtual environment using Nintendo Wii golf putting had not been reported before. Thus, the purpose of the study was to investigate the differences of cortical activity pattern related to sensory information processing during a complex sports-related motor task. On the basis of the literature [5] it was hypothesized that there would be higher values in frontal Theta and parietal Alpha- 2 activity when comparing a putting performance on a real green to a virtual environment, because higher Theta power

and less cortical activity in the parietal cortex seemed to be associated with skilled performance (in this case the golf putt in the real environment).

A total of 10 volunteers ( $26.0 \pm 0.7$  years;  $81.8 \pm 5.6$  kg;  $184.5 \pm 6.0$  cm; handicap  $30.0 \pm 10.0$ ;  $2.9 \pm 1.0$  years of golf experience) participated in the present study and had no history of neurological, cardiovascular or other major disorders, no current use of medications or drugs and no physical or psychological exposures (e.g. intensive training sessions, examinations) 24 hours prior to the trial. All subjects had normal or corrected to normal vision. The study was done in accordance with the rules and regulations established by the Institutional Review Board for ethical treatment of human subjects. All subjects signed an informed consent after the explanation of the testing procedure. All participant data were treated with confidentiality.

The study was conducted at a laboratory at the Institute of Sports Medicine (University of Paderborn). In a preliminary examination the participants were familiarized with the surroundings, the task and procedures one week before the trial. On arriving at the laboratory the electro cap was attached to the participant's head and they completed the health questionnaire. They were asked to lie supine and relax for a 10-minute period. After relaxing, the participants were asked to perform the real or virtual (Wii) putting task in random order. Each performance were followed by a rest period in sitting position (3 min).

The real golf putting took place outside the laboratory where the participants were required to putt golf balls (Bridgestone, USA) on a real golf green towards a target hole (3 m distance) for 3 minutes. In the virtual condition the participants were instructed to play the putting task in a Wii golf game (*Tiger Woods PGA Tour 08*) with a standardized distance with no slopes matched to the real putting situation.

In both tasks they were instructed to do this as accurately as possible at their own pace. During this performance the score was categorized and the EEG signals were recorded.

Performance was categorized in circles around the hole with different scores depending on the proximity to the target hole with lowest score = 0 (distance > 0.8m) and the highest score

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= 15 (categories 1-5: hit [15], 0-0,2m [10], 0.2-0.4m [5], 0.4-0.6m [1], >0.8m [0]). The sum of all putt scores was divided by the number of putts for both conditions.

The EEG was recorded by a stretchable electro cap (ElectroCap Inc., USA) in accordance with standards of the international 10:20 system [14]. During the task performance (3 min) EEG data was collected continuously from frontal (Fz, F3, F4) and parietal (Pz, P3, P4) scalp locations using Cz as a common average reference [15] and a midforehead placement of the ground electrode [20]. All EEG data was recorded and stored using the CATEEM system (MediSyst, Germany). The signals were sampled at 512 Hz / 12 bit and amplified (DC=20 M $\Omega$ ). Before each measurement an impedance test ensured a sufficient signal to noise ratio. The physiological signals were high pass filtered at 0.86 Hz. Due to the problem of eye/head movements automatic artefact detection (depending on artefact rejection limit with respect to the EEG signal amplitude and slew rate limit with respect to the slew rate of the EEG signal [23]) was completed followed by a visual inspection by experienced electroencephalographers. Only artefact-free segments were used for analysis.

In the EEG signals, Fast Fourier Transforms (FFT) was calculated with 50% overlapped, 512 sample Hanning windows for all artefact free segments. Mean absolute EEG spectral power was calculated and divided into three different frequencies: Theta (4.75 – 6.75 Hz), Alpha-1 (7.5 – 9.5) and Alpha 2 (9.75-12.5 Hz). For the statistical analysis, a logarithmic (log) transformation of the power values was used to stabilize variances across subjects [20]. The transformed data in the different frequency bands were computed across both conditions (performance/rest) in each subject.

For statistical analysis SPSS 14.0G software was used. All results are given as mean values and standard deviation. The Kolmogorov-Smirnov (K-S) test was used to determine if variables fit the Gaussian distribution. To examine cortical activity in different frequencies an ANOVA with repeated measures with factors ENVIRONMENT (real vs. virtual) and CONDITION (performance vs. rest) was conducted. Compound symmetry, or sphericity, was



verified by the Mauchley test. When the assumption of sphericity was not met, the significance of F-ratios was adjusted according to the Greenhouse-Geisser procedure. If ANOVA demonstrate a significant interaction effect (ENVIRONMENT x CONDITION), an environmental effect was accepted and pairwise t-tests were made post hoc using a stepwise Holm-Bonferroni adjustment in order to control for Type I error. The outcome of statistical calculations were declared significant if  $p \leq 0.05$ . Partial  $\eta^2$  were computed to measure the effect sizes, which ranged from small (0.01 – 0.05) to medium (0.06 – 0.13) to large ( $\geq 0.14$ ) [6]. In case of significances in post hoc tests effect sizes were calculated according to Cohen's d. Here an effect size of 0.20 implied a small effect, 0.50 a medium effect, and 0.80 a large effect.

The participants achieved significantly higher scores during the real (average number of putts:  $M=12.5$ ,  $SD=2.1$ ; score:  $M=8.8$ ,  $SD=2.2$ ) compared to the virtual putting (average number of putts:  $M=11.8$ ,  $SD=1.4$ ; score:  $M=5.5$ ,  $SD=2.3$ ) situation ( $T(9)=3.306$ ,  $p=0.009$ ;  $d=1.45$ ) in a 3 min performance.

Putting in the virtual environment led to differences compared to the real condition in EEG power related to the working memory in the prominent Theta and Alpha-2 frequency bands. Additionally there were no further significant effects (ENVIRONMENT  $F_{1,9}=0.001-2.388$ ;  $p=0.157-0.974$ ; CONDITION  $F_{1,9}=0.039-2.321$ ;  $p=0.162-0.911$ ) in the Alpha-1 spectral power in frontal and parietal brain areas.

Whereas the statistical analysis for Theta spectral power revealed no main effect for ENVIRONMENT the factor CONDITION shows significantly higher values during performance compared to rest at all electrode positions ( $F_{1,9}=12.137-29.284$ ;  $p=0.001-0.007$ ; partial  $\eta^2=0.574-0.765$ ). Furthermore Theta power demonstrates an interaction effect (ENVIRONMENT x CONDITION) at Fz ( $F_{1,9}=5.808$ ;  $p=0.039$ ; partial  $\eta^2=.392$ ), F3 ( $F_{1,9}=7.932$ ;  $p=0.020$ ; partial  $\eta^2=0.468$ ) and F4 ( $F_{1,9}=7.007$ ;  $p=0.027$ ; partial  $\eta^2=0.438$ ). Post hoc analysis revealed significant higher values in real compared to virtual performance (ENVIRONMENT) at F3 ( $T(9)=2.617$ ;  $p=0.046$ ;  $d=0.66$ ; Fig.1) and F4 ( $T(9)=2.941$ ,  $p=0.032$ ;  $d=0.69$ ; Fig.1) and an activation effect (CONDITION) with significant higher spectral power in performance in

both conditions compared to rest at electrode positions Fz (real:  $T(9)=5.817$ ,  $p=0.001$ ;  $d=1.10$ ), F3 (real:  $T(9)=4.918$ ;  $p=0.004$ ;  $d=1.53$ ; virtual:  $T(9)=4.312$ ;  $p=0.006$ ;  $d=0.92$ ) and F4 (real:  $T(9)=5.005$ ,  $p=0.004$ ;  $d=1.40$ ; virtual:  $T(9)=3.039$ ,  $p=0.048$ ;  $d=0.84$ ; Fig 1).

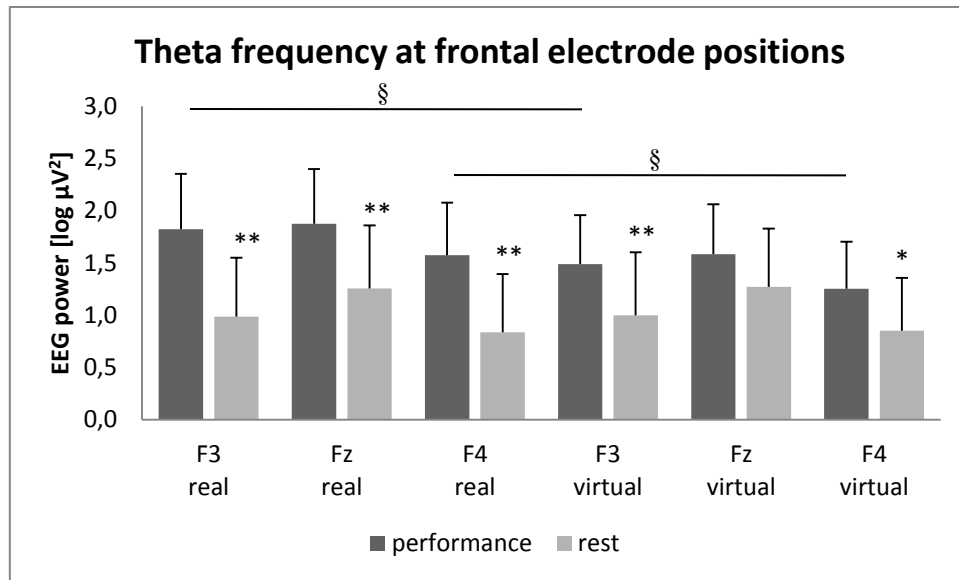


Figure 1: EEG spectral power [ $\log \mu V^2$ ] during real and virtual putting (performance / rest) in frontal Theta frequency

(\*  $p \leq 0.05$ , \*\*  $p \leq 0.01$ : significance level (Holm-Bonferroni adjusted) according to factor CONDITION between performance and rest in each environment; §  $p \leq 0.05$ ; significance level (Holm-Bonferroni adjusted) according to factor ENVIRONMENT between real and virtual in performance and rest)

Alpha-2 power shows no main effect according to ENVIRONMENT but CONDITION revealed an effect at electrode positions Pz, P3 and P4 ( $F_{1,9}=5.793-9.714$ ;  $p=0.012-0.039$ ; partial  $\eta^2=0.392-0.519$ ). Additionally the participants performed with an interaction effect (ENVIRONMENT x CONDITION) in the Alpha-2 frequency band at electrode position P3 ( $F_{1,9}=7.688$ ;  $p=0.022$ ; partial  $\eta^2=0.461$ ). Further analysis demonstrate a higher Alpha-2 spectral power in real vs. virtual (ENVIRONMENT) during the performance at P3 ( $T(9)=2.345$ ;  $p=0.044$ ;  $d=0.47$ ; Fig 2) and increased values in the virtual condition during performance compared to rest (CONDITION) at the parietal position P3 ( $T(9)=3.513$ ;

$p=0.028$ ;  $d=0.84$ ; Fig.2). Parietal Alpha-2 spectral power demonstrated no significant interaction effects at electrode positions P4 ( $F_{1,9}=4.677$ ;  $p=.059$ ) and Pz ( $F_{1,9}=0.445$ ;  $p=.522$ ).

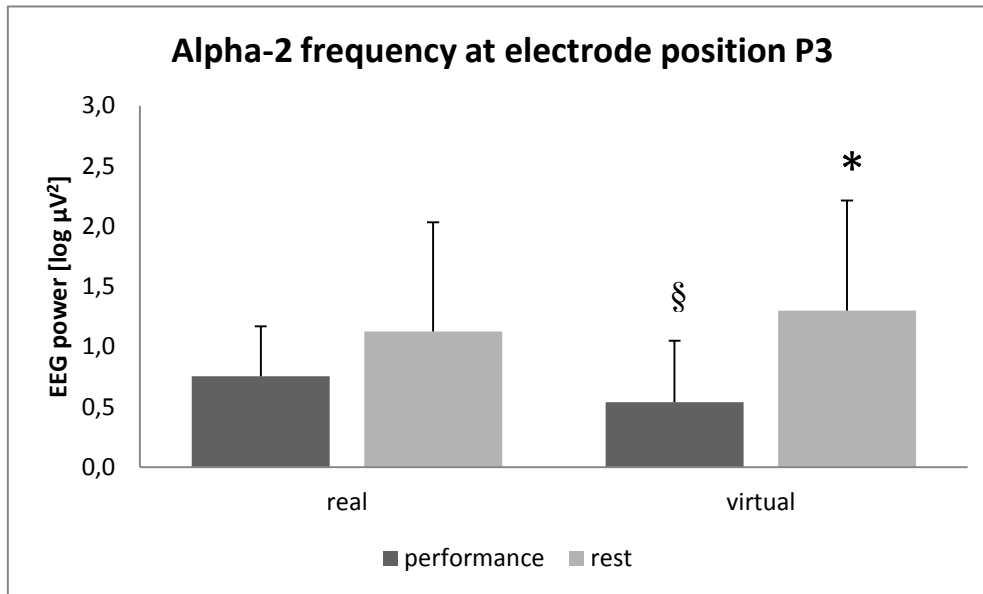


Figure 2: EEG spectral power [log μV<sup>2</sup>] during real and virtual putting (performance / rest) in parietal Alpha-2 frequency

(\*  $p \leq 0.05$ : significance level (Holm-Bonferroni adjusted) according to factor load between performance and rest in each condition; §  $p \leq 0.05$ : significance level (Holm-Bonferroni adjusted) according to factor condition between real and virtual in performance and rest)

The cortical activation pattern demonstrated differences in frontal Theta power and Alpha-2 spectral values when comparing a putting performance in a real and a virtual environment using the Nintendo Wii. In contrast Alpha-1 spectral power was not affected. Usually different environmental conditions due to light led to a decrease of Alpha-1 power especially in parieto-occipital brain areas which would affect the results in this study and might have led to an interpretation bias. But in this case unaffected Alpha-1 power indicated that different light conditions (real putting performed outdoors and virtual putting indoors) had not influenced the results.

All participants had experience in playing golf and had been engaged in the putting task. However, the score show differences between both conditions with higher scores obtained in

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the real task. That may be due to the fact that the subjects were used to this environment and that the virtual putting was a new task for them.

This result was accompanied by changes in the prominent EEG power frequencies related to the working memory. Frontal Theta power was able to distinguish between the environment and conditions with higher values in the real putting as well as in performance compared to rest. This finding was consistent with numerous studies indicating the role of this frequency associated with attention in cognitive [7], sensorimotor [24,4] and sports related tasks [5, 8]. Different approaches suggested the anterior cingulate cortex (ACC) as the generator of frontal Theta frequency. Gevins et al. [9] used EEG in conjunction with Magnetic Resonance Imaging (MRI) and located the medial prefrontal structures in the region of the ACC as the source of Theta frequency. This was supported by Onton et al. [19] who used a dipole source model whereas Sauseng et al. [22] located frontal Theta generated by the ACC during a sensorimotor task by using the LORETA approach. The ACC was involved in a range of executive functions such as processing information and decision making but most investigators connected this subcortical region with an important component of the human attentional control system [16] which was involved in working memory processing.

In the current study the participants performed with increased frontal Theta power in the putting performance compared to the resting period in both conditions. The most often reported relationship demonstrated an increase in the amount of frontal Theta with increasing working memory load, task difficulty and mental effort which was described as closely related to focussed attention [9, 12, 25]. Following this idea the frontal Theta power was able to discriminate between performance and rest. Furthermore the results demonstrated significant differences between the real and the virtual putting condition. Based on the increased Theta power this might reflect a higher focussed attention in the real condition. Speculations may lead to the idea that the participants performed a task which they were used to and where they knew the level of concentration which was needed to select the relevant sensory information for a good performance [5]. On the other hand it might be speculated that in terms of the novelty of the virtual putting task the subjects might have no idea what

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information was relevant in this condition and therefore might have no basis for attention and decision making. This might be an explanation for the significant lower Theta power in virtual putting. Additionally this was supported by the significant lower score in the Wii condition compared to the real putting. Häufner et al. [13] supported the results and had found higher frontal Theta power in marksmen compared to novice shooters during a shooting task. They explained their findings with the expert's familiarity with the task and the concentration on the tasks central demands opposite to the novices.

The second prominent frequency in the working memory model was the fast Alpha frequency. Alpha activity was often described as a form of cortical idling with its amplitude inversely related to the number of neuronal populations activated during cognitive and motor processes [18]. The fast Alpha (Alpha-2) tended to be attenuated by task-specific demands depending on parieto-occipital regions in the area of the somatosensory cortex. The higher Alpha-2 parietal (P3) power in the real condition implied less cortical activation in the parietal regions compared to Wii putting. That meant the participants must actively process unfamiliar cues in the virtual environment that seemed to require more neuronal resources in the parietal cortex. These findings were consistent with the results of Gevins et al. [9] who had described practice-related increases in Alpha-2 power with fewer cortical resources involved after skill development in a cognitive n-back task. Similar results were described for visuomotor tasks [25]. Baumeister et al. [5] demonstrated enhanced Alpha-2 power values in conjunction with higher expertise in golf putting task. They suggested that the development of task-specific expertise might be reflected in an increased Alpha-2 power and therefore less cortical activity in the somatosensory cortex.

Nevertheless two limitations should be considered when drawing interpretations from these findings. First, we had not controlled the movement. Even if we tried to instruct the participants to do the same movement in real and virtual environment and subjectively controlled it by observation it there is no objective data base to compare both movements. Further studies should add a motion analysis system to quantify the movements. Secondly, the study population was relatively small. Especially the statistical power for detecting further

effects in the parietal Alpha-2 frequency might affect the results. Larger studies were needed to increase statistical power and confirm the changes in the somatosensory cortex related to real and virtual environments.

To conclude the results of this study demonstrate that there was a difference in cortical activity pattern related to the working memory during a golf putting task in a real and a virtual environment using Nintendo Wii. The results demonstrated an increase in frontal Theta due to higher attention and parietal Alpha-2 power related to less cortical activity in the somatosensory cortex in the real golf putting. Based on the cortical activity obtained in this study a virtual golf putting training might not be able to replace a training session in a real environment. Further studies should focus on transfer effects from virtual to real environments to give a suggestion if virtual environments can be used in golf training.

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## PUBLICATION 6

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<b><i>Analysis and interpretation</i></b>	<b>Reinecke, Baumeister, Weiss</b>
<b><i>Drafting the manuscript</i></b>	<b>Reinecke, Baumeister</b>
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## ABSTRACT

Although neurophysiological aspects have become more important in sports and exercise sciences in the last years, it was not possible to measure cortical activity during performance outside a laboratory due to equipment limits or movement artifacts in particular. With this pilot study we want to investigate whether EEG data obtained in a laboratory golf putting performance differ from a suitable putting task under field conditions. Therefore, parameters of the working memory (frontal Theta and parietal Alpha 2 power) were recorded during these two conditions. Statistical calculations demonstrated a significant difference only for Theta power at F4 regarding the two putting conditions "field" and "laboratory". These findings support the idea that brain activity patterns obtained under laboratory conditions are comparable but not equivalent to those obtained under field conditions. Additionally, we were able to show that the EEG methodology seems to be a reliable tool to observe brain activity under field conditions in a golf putting task. However, considering the still existing problems of movement artifacts during EEG measurements, eligible sports and exercises are limited to those being relatively motionless during execution. Further studies are needed to confirm these pilot results.

**Key Words** EEG, laboratory, field, golf putting, working memory

## INTRODUCTION

Neurophysiological aspects of goal-directed movements, motor learning and skilled performance in sports and exercise sciences have become more important during the past years. Especially the brain as the central motor of movement planning and control is currently a research subject with increasing interest to bridge the gap between behavior related research in sports sciences and neuroscientific models of basic mechanisms that support sporting performance (Kubesch 2004; Jansen-Osmann 2008; Yarrow et al. 2009). Electroencephalography (EEG) represents a methodological tool to display brain activity before, during and after cognitive and motor performance, caused in its excellent temporal resolution and the advantages of wireless hardware as well as equipment portability (Thompson et al. 2008). Besides archery (Salazar et al 1990) and rifle or pistol marksmanship (Häufner et al 2000; Hatfield 1984; Hillman 2000), golf putting seems to be an adequate sports performance to evaluate EEG based neurocognitive processes depending on skill level or skill acquisition. Baumeister et al. (2008a) were able to distinguish expert and novice golfers regarding the load of working memory during a putting task. The experts demonstrated higher frontal Theta Power values and higher parietal Alpha-2 power values compared to the novice golfers. In the concept of working memory, this was suggested to represent a state of higher focused attention (frontal Theta) and an economy of effort in sensory information processing (parietal Alpha-2). Furthermore, Crews and Landers (1993) investigated neuronal correlates of attentional patterns prior to the golf putt in highly skilled golfers. Babiloni et al. (2008) detected a relation between successful putts and high-frequency Alpha power in the frontal midline and the right sensorimotor brain area. These neurocognitive patterns were pointed to be predictive for golfer's performance. Those investigations are providing reliable data concerning cortical activation patterns during a complex sports-related motor task (golf putting). However, they all took place under laboratory conditions. Investigations accomplished in a lab can be considered as well-controlled and reproducible (Riley et al. 2008). But several sports and exercise studies suggest that laboratory based findings do not automatically hold in training and competition

situations under field conditions. Riley et al. (2008) compared kinematic and kinetic parameters of treadmill and overground running. Their results were comparable but not directly equivalent. In addition, it has been shown by Meyer et al. (2003) and Di Michele et al. (2009) that identical running protocols on different surfaces like treadmill, track, natural grass or synthetic turf cause varying physiological responses. These findings suggest that data from laboratory examinations are similar to field conditions but can not be transferred unevaluated. What has been demonstrated in the research field of sports and exercise related to training and testing as well as biomechanics lacks in the neuroscientific domain. Hence, it appears to be necessary to investigate whether neurocognitive patterns measured under laboratory conditions are transferable into field conditions and if the EEG tool is able to deliver comparable and therefore reliable data in a real sports environment likewise.

In this context the overall aim of the present pilot study was to compare brain activity results from laboratory and field conditions using working memory functions during golf putting as a model. We hypothesized that (1) the EEG data obtained during laboratory golf putting conditions do not significantly differ compared to suitable tasks under field conditions. Furthermore, this should be supplemented by previously described results (Baumeister 2008a) which indicate that (2) working memory functions differ between golf putting performance and a corresponding resting situation. This pattern is hypothesized to be reproducible both in the field and the laboratory condition to confirm a basically stable testing situation.

## **METHODS**

### **Subjects**

Eleven male students from the University of Paderborn ( $25.6 \pm 0.8$  years;  $80.9 \pm 5.4$  kg;  $185.3 \pm 6.7$  cm) volunteered in this study. They were recruited from the affiliated golf club. The participants had experiences in playing golf ( $3.5 \pm 2.4$  years playing golf; playing golf  $4.6 \pm 3.5$  hours per week) and a mean golf handicap of  $28.4 \pm 11.9$ . All subjects were

categorized as right-handed evaluated with the EHI questionnaire (Oldfield 1971). Additionally, participants completed a health questionnaire to avoid any historical or current existence of neurological, cardiovascular or other major disorders which could influence EEG results. They showed no current use of medications or drugs and no physical or physiological exposures (i.e. intensive training sessions, examinations) 24 hours prior to the main trial. All participants had self-reported normal or corrected to normal vision at the time of the experiment. The study was done in accordance with the rules and regulations established by the Institutional Review Board for ethical treatment of human subjects. All participants signed an informed consent after the explanation of the testing procedure. All data were treated with confidentiality.

## **Procedures**

The study was conducted in a laboratory at the University of Paderborn. The participants were familiarized with the surroundings, the tasks and the procedures in a preliminary examination one week in advance of the main trial. The electrode cap (ElectroCap Inc., USA) was explained and attached and after introducing the two putting conditions they were asked to execute both with a corresponding resting period to get used to the experimental protocol. In addition, they were in particular trained to stay as relaxed as possible with their facial muscles in order to reduce artifacts. Afterwards the health and EHI questionnaires were completed. EEG was not measured during this session.

After arriving to the main trial of the study the electrode cap was attached to the participants' head. They were asked to fill in the STAI X1 to evaluate their current emotional status with respect to feelings like anxiety, tension and nervousness. This was followed by a relaxing period of 10 minutes in a supine position. After relaxing they were required to putt golf balls (Bridgestone, USA) in two different conditions for two minutes each. In the laboratory putting condition a green carpet (ENIA Carpet, Germany; stimp meter rate: 9—mean comparable speed to medium fast greens) was used as performing surface. Disturbances caused by

noise or varying lighting conditions could be excluded in the laboratory condition. The putting task in the field condition took place on an ordinary putting green without slopes on a golf course directly outside the laboratory. Before the outdoor measurement was executed the participants got the chance to adjust themselves to the different lightning and noise conditions. This was done to reduce the occurrence of artifacts in the EEG parameters because of eye blinks, for example.

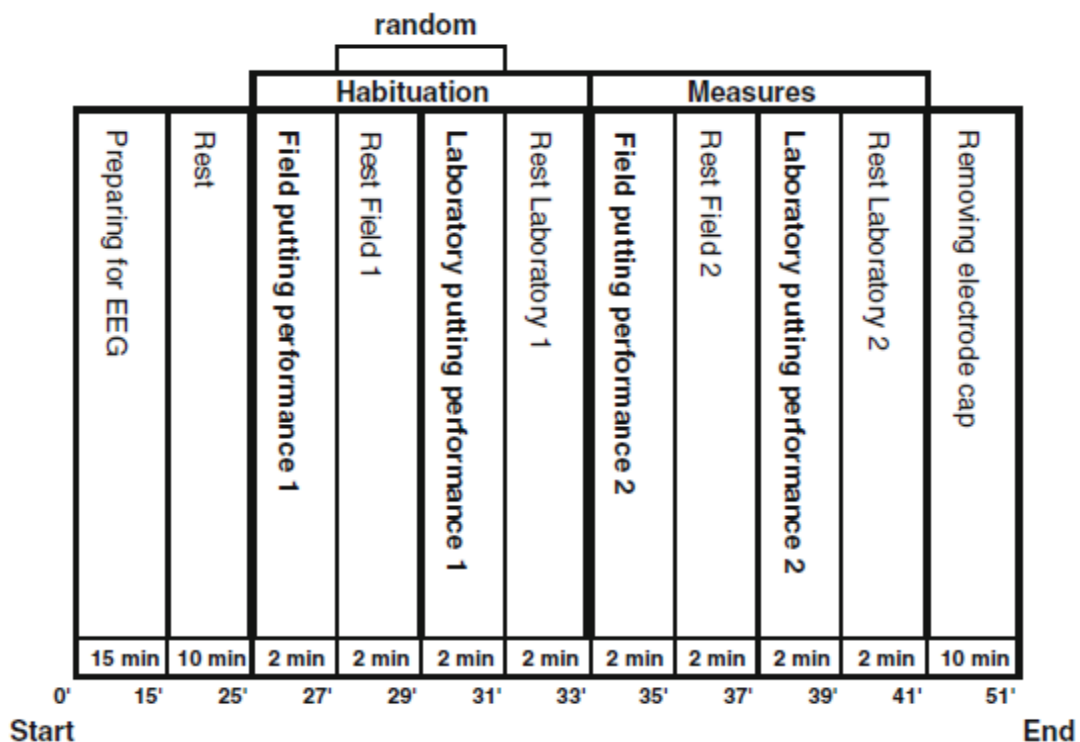


FIGURE 1. Study design

The participants had to putt balls into a real hole whereas the target on the carpet in the laboratory represented real hole size. The subjects were instructed to perform their putting from a 3m distance as accurate as possible with at their own pace. For performing they used a standardized putter (UG-LE, Ping, USA). No practice putts were allowed. There was no verbal feedback given to the participants but they had their visual feedback after each stroke. Each putting condition was alternately executed twice (heat 1/heat 2) whereas the starting condition was randomized (Fig.1). Every putting performance was followed by a corresponding resting period which also lasted two minutes and was arranged in a

standardized sitting position. EEG was recorded continuously during performance and resting periods but only the particular second heat of the two conditions (performance and rest) was analyzed due to a habituation process. Therefore, participants wore a small backpack which contained the EEG amplifier. A 10m optical fiber wire was connected with the main computer to assure mobility for both the laboratory and the field putting task. Subjective stress level was requested after each performance situation. The participants completed the STAI X1 once again in the end of the trial.

## **Measurements**

Psychometric measures. State anxiety was assessed using the Spielberger State–Trait Anxiety Inventory (Laux et al. 1981). The state anxiety scale (STAI X1) includes 20 items that evaluate current feelings of tension, anxiety and nervousness. Cumulative values can range between 20 and 80, in which 20 represents an absence and 80 a maximal intensity of the described feelings. The STAI is a reliable and valid measure that has been used with both clinical and nonclinical populations (Laux et al. 1981; Quek et al. 2004).

The subjective stress level was measured using a visual analogue scale (VAS) (Baumeister et al. 2008a and 2008b). Immediately after every putting performance participants were asked to state their individual level of stress on a scale-line between „no stress at all“ (0 cm) and „highest stress level“ (10 cm) respectively.

Electroencephalography. All EEG data were recorded and stored using the CATEEM system (MediSyst, Linden, Germany). In accordance with the international 10:20 system (Jasper 1958) the recordings were accomplished by a stretchable electrode cap (ElectroCap Inc., USA). During the two task performances and the corresponding resting periods (each lasting 2 minutes) EEG was recorded continuously from 17 scalp locations (Fz, F3, F4, F7, F8, Cz, C3, C4, Pz, P3, P4, T3, T4, T5, T6, O1, O2) using a common average reference montage (Lehmann 1987; Baumeister et al. 2008a, 2008b, 2011) and a midforehead placement of the ground electrode (Pivik et al. 1993). Due to movement artifacts electrode positions F7, F8,

O1 and O2 were excluded from analysis a priori. The signals were sampled at 512 Hz / 12 bit and amplified (DC=20 M $\Omega$ ). An impedance test (< 50 k $\Omega$ ) ensured a sufficient signal to noise ratio before each measurement (Schober et al. 1995). The physiological signals were highpass filtered at 0.86 Hz. For identification and elimination of artifacts interactively the following criteria were used: the oculogram and automatic artifact rejection limit was defined with respect to the EEG signal amplitude and slew rate limit with respect to the slew rate limit of the EEG signal (Schober et al. 1995). This was visually controlled offline by experienced electroencephalographers. Only artifact-free segments were used for analysis. During both the outside and the laboratory putting condition more than 80% and during resting more than 93% of the EEG data could be used for analysis. In the EEG signals, Fast Fourier Transforms (FFT) were calculated on 50% overlapped, 512 sample Hanning windows for all artifact free segments with the CATEEM system. The power spectra were divided into different frequencies: Theta (4.75 – 6.75 Hz), Alpha-1 (7.0 – 9.5 Hz), Alpha-2 (9.75-12.5 Hz) and Beta 1 (12.75 – 18.5 Hz). Data were computed across all heats for performance and resting periods for each participant. For statistical analysis, EEG power values were log-transformed to stabilize variances across subjects (Pivik et al. 1993). In this case statistical analysis of the central parameters were restricted to working memory functions in the fronto-parietal network (Sauseng et al. 2005). Therefore, only the Theta frequency band for frontal electrode positions (Fz, F3, F4) and the Alpha-2 frequency band for parietal electrode positions (Pz, P3, P4) were included into analysis.

### **Statistical Analysis**

SPSS 19.0 software was used for statistical analysis. Results are given as mean values and standard deviation. The Kolmogorov-Smirnov (K-S) test was applied to determine if variables fit the Gaussian distribution. To examine differences in cortical activity, a 2-way ANOVA was conducted (factor CONDITION: Field – Lab; factor LOAD: performance – rest) for EEG parameters. Effect sizes were calculated using the Partial  $\eta^2$  (small: 0.01 - 0.05; medium: 0.06 - 0.13; large:  $\geq$  0.14; Cohen 1988). In case of main or interaction effects, paired t-tests

were used post-hoc. If significances in t-tests occurred, Pearson's correlation coefficient,  $r$ , was calculated to provide effect sizes (Rosnow et al. 2000). Here,  $r = 0.20$  implies a small effect,  $r = 0.50$  a medium effect and  $r = 0.80$  a large effect. To evaluate differences regarding the psychometric measurements (STAI X1: before – afterwards; subjective stress level: field - laboratory) paired t-tests were made. Overall significance level was set at  $p \leq 0.05$ .

## RESULTS

### Psychometric measures

The state anxiety scores from the beginning ( $32.7 \pm 6.6$ ) and the end ( $33.7 \pm 7.9$ ) of the trial demonstrated no significant difference ( $p=0.365$ ).

The subjective stress level was slightly higher after the laboratory performance ( $1.7 \pm 1.2$ ) compared to the field performance ( $1.3 \pm 1.1$ ). However, that difference did not reach statistical significance ( $p=0.118$ ).

### EEG parameters

A comparison of the selected EEG parameters revealed significant differences in the frontal Theta power values only. These differences mainly occurred with respect to factor LOAD. In more detail, frontal Theta and parietal Alpha-2 mean values, standard deviations and ANOVA results (CONDITION: Field and Lab; LOAD: performance and rest) are displayed in table 1. Significant post hoc findings are presented below.

#### Differences between Field and Laboratory

ANOVA demonstrated a significant main effect (factor CONDITION) for Theta at electrode position F4 only (table 1). Post hoc t-tests confirmed the slightly higher power values during the field performance situation compared to the laboratory putting task ( $t(10) = 3.037$ ;  $p \leq 0.02$ ;  $r = 0.69$ ). Occurring interaction effects (CONDITION x LOAD) were tested with regards



to CONDITION differences post-hoc. Here, in addition to results for Theta power at F4, significant performance differences for Theta power at F3 could be detected ( $t(10) = 3.017$ ;  $p \leq 0.02$ ;  $r = 0.69$ ). No significant differences emerged in the Alpha-2 frequency band.

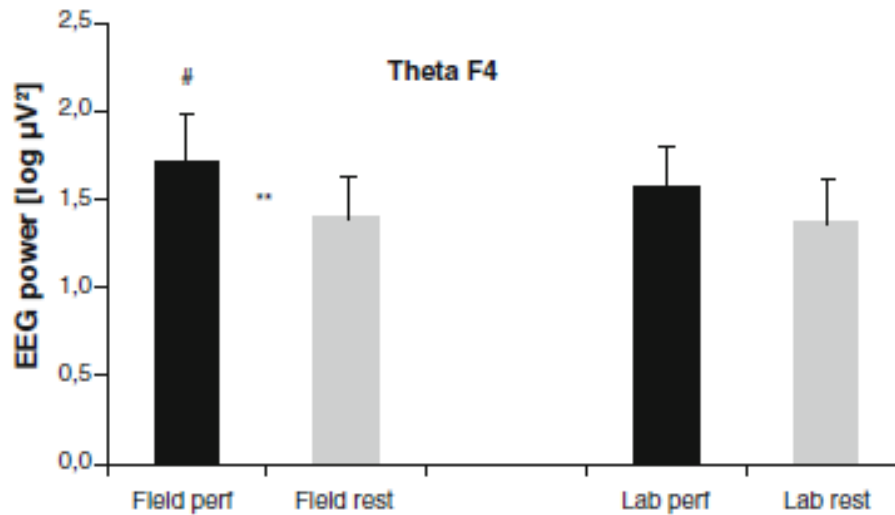


FIGURE 2. EEG spectral power for Theta F4 (significances: \*\* factor LOAD:  $p \leq 0.01$ ; factor CONDITION:  $p \leq 0.05$ )

#### Differences between Performance and Rest

Frontal Theta power values were basically higher during performance compared to rest, which became manifest in ANOVA main effects (factor LOAD) (table 1). The field condition post hoc t- tests significantly confirmed these differences at Fz ( $t(10) = 3.358$ ;  $p \leq 0.01$ ;  $r = 0.73$ ), F3 ( $t(10) = 3.601$ ;  $p \leq 0.01$ ;  $r = 0.75$ ) and F4 ( $t(10) = 3.164$ ;  $p \leq 0.01$ ;  $r = 0.71$ ). In the laboratory environment differences between performance and rest became significant at Fz ( $t(10) = 2.891$ ;  $p \leq 0.02$ ;  $r = 0.67$ ) and F3 ( $t(10) = 3.320$ ;  $p \leq 0.01$ ;  $r = 0.72$ ). In contrast parietal Alpha-2 demonstrated higher power values during the resting period in both the field and the laboratory condition, which did not reach significance.

CONDITION	Field		Laboratory		ANOVA		
	Performance	Rest	Performance	Rest	CONDITION	LOAD	INTERACTION
Theta Fz	1.84 ± 0.25	1.56 ± 0.27	1.78 ± 0.25	1.54 ± 0.27		F <sub>1,10</sub> = 10.995; p = 0.008; ES = 0.524	
Theta F3	1.81 ± 0.28	1.43 ± 0.31	1.66 ± 0.23	1.45 ± 0.27		F <sub>1,10</sub> = 12.995; p = 0.005; ES = 0.565	F <sub>1,10</sub> = 8.167; p = 0.017; ES = 0.450
Theta F4	1.72 ± 0.28	1.39 ± 0.24	1.56 ± 0.24	1.37 ± 0.25	F <sub>1,10</sub> = 5.230; p = 0.045; ES = 0.343	F <sub>1,10</sub> = 7.433; p = 0.021; ES = 0.426	F <sub>1,10</sub> = 21.305; p = 0.001; ES = 0.681
Alpha 2 Pz	1.34 ± 0.23	1.56 ± 0.54	1.29 ± 0.23	1.66 ± 0.55			
Alpha 2 P3	1.34 ± 0.17	1.52 ± 0.42	1.32 ± 0.20	1.56 ± 0.44			
Alpha 2 P4	1.31 ± 1.15	1.55 ± 0.56	1.30 ± 0.19	1.64 ± 0.53			

TABLE 1. EEG power spectra [log  $\mu\text{V}^2$ ] ( $M \pm SD$ ) and ANOVA results for Theta (F3, Fz, F4) and Alpha-2 (P3, Pz, P4) during putting performance and rest field and laboratory conditions

## **DISCUSSION**

### **Psychometric measures**

The low and non-significant subjective stress values after the laboratory and the putting condition in the field suggest that the participants felt comfortable during the whole experimental situation. The same applies to the state anxiety scores before and after the trial. Hence, we assume that the subject's psychological state remained constant during the investigation.

### **EEG parameters**

#### Differences between Field and Laboratory

The two putting conditions can be characterized as comparable. Both frontal Theta and parietal Alpha-2 power values between the laboratory and the field condition during performance as well as during rest appear to be similar but not exactly equivalent. This goes along with findings from former lab-field comparisons in other areas of sports and exercise (Riley et al. 2008; Meyer et al. 2003; Di Michele et al. 2009). It may indicate that on the one hand the cognitive load in laboratory tasks seems to be related to field conditions but also calls for survey in field environments to exactly determine cortical activation patterns. Disturbances like noise or lightning conditions may alter cognitive demands and that needs to be addressed. On the other hand we were able to demonstrate for the first time that the EEG methodology appears to be a reliable tool to map cortical activation patterns under field conditions during golf putting.

#### Differences between Performance and Rest

The testing situation can be characterized as stable. Both Theta and Alpha-2 power values can be distinguished between performance and resting in the laboratory as well as the field condition. In general, EEG frontal Theta and parietal Alpha-2 power are connected with working memory functions: frontal Theta power is related to the attentional system and

parietal Alpha-2 power is referenced to somatosensory information processing (Sauseng et al. 2005). Former investigations reported increased Theta power during cognitive, visuomotor and sensorimotor performances (Baumeister et al 2008a and 2008b; Grunwald et al. 2001; Slobounov et al. 2000; Smith et al. 1999), which was interpreted with increased attentional demands during those specific tasks. Following these conclusions, the significantly higher frontal Theta power values during performance compared to the resting period in the present study might reflect the participant's engagement in the putting task in contrast to a "cognitively neutral" (Hatfield et al. 1984) state during resting.

The second prominent frequency in the concept of working memory, parietal Alpha-2, represents task-specific aspects of sensory information processing (Gevins et al. 1997; Smith et al. 1999). The Alpha frequency amplitude is inversely related to the numbers of activated neuronal populations (Niedermeyer and Lopes da Silva 2005). In this study the decreased Alpha-2 power values during performance compared to rest seem to appear in accordance with already existing findings (Niedermeyer and Lopes da Silva 2005). This might represent a state of higher sensory information processing during putting and a state of less cortical activation during resting. However, the observed differences did not reach significance due to high individual differences during the resting period (Baumeister et al. 2011). Inter- and intra-individual Alpha differences are a well-known and intensively investigated phenomenon (Shaw 2003). For example, different head sizes and ages were reported to be responsible for varying inter-individual Alpha rhythms (Nunez et al. 1978). Also intra-individual changes in the Alpha rhythm depending on cognitive and task demands have been found (Klimesch et al. 1993). This could be an explanation for the widespread variation of Alpha 2 values during the mentally unspecific brain state while resting in the present study whereas the subject's cognition during performance was directed to the putting task. A standardization of those resting periods can probably help to minimize variations in the Alpha frequency.

We are aware of the fact that these results are based on a pilot study including only one measurement per condition (laboratory-field) and situation (performance-rest) and a relatively

small number of participants. Although we established a stable testing situation, a future challenge will be to approve these results in similar investigations with more repetitions in each situation and to analyze the remaining electrode positions and brain areas. Alternative analytic approaches should also be considered in future investigations: Equivalence testing represents an adequate statistical tool for studies like ours with the intent to show no differences. A more expanded ANOVA model would also be an option to reveal differences in EEG parameters. If we manage to strengthen the present results, several possibilities for future research regarding neurocognitive processes in goal-directed sports performances appear: Provided that equipment is both portable and wireless and the selected motor performance is appropriate due to movement artifacts and limits, skill acquisition processes could be observed under field conditions. It would then be possible to not only include movement outcome and technique analysis as tools for skill development. Additionally neurocognitive processes which have been proofed to be linked to skill improvement could be considered as well (Gevins et al. 1997; Smith et al. 1999). This might be helpful to more precisely control skill acquisition processes and interfere when athletes fail during motor learning (Yarrow et al. 2009). Another possibility could be the online monitoring of working memory load in athletes during training sessions. If increased performance error in a goal-directed sports performance is a consequence of mental overstraining and fatigue development, the brain would be the first place to examine evidence for upcoming performance failures.

## **SUMMARY**

In conclusion the results of the current pilot study provide first indications to extend the limitations of the EEG methodology in the application in sports and exercise sciences with. The load of working memory appears to be similar during laboratory and field conditions in a goal-directed motor task. Our findings may offer several possibilities for future research including analysis of neurocognitive processes during skill acquisition and training processes under field conditions. However, due to the pilot character of the present investigation

methodological interpretations have to be considered carefully and further studies are needed to confirm these first results.

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## CHAPTER 8

### **Sensorimotor Control and Brain Activity in injury-related conditions**

Rehabilitation programs in musculoskeletal disorders focus mainly on the muscle to restore functions. Although the methodological approach is difficult, it is important to know how the brain regulates sensorimotor control in order to understand the neuronal mechanisms on which sensorimotor control is based on after musculoskeletal damage.

Three papers in this research program demonstrate that injury-related conditions resulting from damage to the musculoskeletal system (ACL injury) and pain (exercise induced muscle damage) cause variations in EEG power according to the working-memory-hypothesis in different somatosensory modalities.

This Chapter is introduced by a publication describing that musculoskeletal rehabilitation can greatly benefit from neuroscientific knowledge of brain activity associated with peripheral injuries ([Publication 7](#)). For future research in sensorimotor control related musculoskeletal rehabilitation the importance to open the window to the brain should be considered and an interdisciplinary approach is needed to discover the underlying mechanisms behind injury and damage.

ACL injuries are a good deficit model for musculoskeletal disorders, because it can be assumed that there is a deficit in proprioceptive afferent information ([Publication 8](#)). It is hypothesized that sensory deficits cause alterations in cortical information processing. Twelve patients after ACL reconstruction carried out a knee joint position reproduction task and were compared to ten healthy controls. During the performance of the task EEG data was obtained and EEG power was calculated. Task performance was decreased in ACL patients indicated by a higher aberration error in the knee angle matching compared to the controls. Associated brain activity demonstrated changes related to the working-memory-hypothesis. Frontal Theta was enhanced and parietal Alpha-2 power was lower in the ACL

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patients indicating higher information processing demands and attentional control as well as higher processing activity in the parietal cortex.

According to the sense of force, a force reproduction task was conducted with nine patients after ACL reconstructions which were compared to healthy controls ([Publication 9](#)). The task required the participants to reproduce 50% of their individual MVC of the non-injured side. The aberration error demonstrated no differences compared to the healthy controls whereas EEG power show higher values in the ACL patients related to higher demands related to the working-memory-hypothesis. Compared to the findings in the joint position sense task the results indicate different activity patterns in ACL patients which might be due to different proprioceptive modalities involved.<sup>10</sup>

The somatosensory modality of pain had been investigated in collaboration with the University of Cape Town, South Africa using an exercise induced muscle damage (EIMD) protocol ([Publication 10](#)). EIMD is a good model for the modality of pain, because damage and pain can be quantified and characterized very precisely. Sixteen subjects participated in an EIMD protocol and were monitored over 132 hours (before and at 12, 26, 60, 84, 108, 132 hours) after inducing damage to the muscle. Twelve subjects serve as a control group. The participants had to conduct an extension/flexion movement of the elbow at every evaluation point in time. The results demonstrate that Alpha-2 power is sensitive to variations in pain after an EIMD protocol. The findings might indicate a suppression of the sensation of pain in perception circuits.

The ACL injury is a good model for injury-related conditions. Damage to the ligament affects the brain state. This indicates that the working memory is sensitive to changes in EEG power due to different proprioceptive modalities. In the next step, a longitudinal study is needed to monitor rehabilitation programs. This knowledge will help to optimize and evaluate therapy programs and use the underlying central mechanism to restore peripheral function.

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<sup>10</sup> A study looking at the sense of motion has been conducted in collaboration with Eva Ageberg in Sweden supported by the DFG (BA 4062/2-1). The results had been analyzed and are currently prepared for publication by Eva Ageberg.

## PUBLICATION 7

Baumeister, J. (2012). What the brain can tell us in musculoskeletal rehabilitation  
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Author Contributions:

***Concept and design:*** Baumeister

***Drafting the manuscript*** Baumeister

***Critical intellectual revision*** Weiss

ABSTRACT

- None -

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Research in musculoskeletal rehabilitation has grown tremendously in the past few decades. Most of this research is based on a biomechanical approach demonstrating peripheral differences between patients and controls in kinetic, kinematic and electromyographic data. The scientific community describes motor behavior as the final common output of the human sensorimotor system which provides valuable insights into motor changes after musculoskeletal injuries and damages.

But the sensorimotor system covers far more than bones and muscles to provide control of motor behavior. Sensorimotor control describes the dynamic interaction between sensations of sensory stimuli (e.g. proprioceptive, visual, vestibular), the processing and integration of these information in the CNS and the biomechanically well described motor output [1].

Undoubtedly the brain is the supervisor of sensorimotor control. It is important to know how the brain regulates the control mechanisms in order to understand how training in rehabilitation should be designed to compensate in motor deficits after injury [2]. Only this brain-behavior relationship can provide the foundation of rehabilitation programs.

The ACL injury seems to be the most investigated musculoskeletal injury and therefore is a good model in this case. There had been extensive research to highlight the deficits like muscle strength [3], postural control [4] and coordination patterns in functional performance [5–7] which occur after ACL injury and/or reconstruction. These deficiencies are thought to be related to the disturbance of the sensorimotor control process [8, 9]. On a theoretical basis different authors hypothesize that the deficiency in motor output is related to the change of the reception of afferent proprioceptive information due to receptor damage in the knee caused by ACL injury or reconstruction which most likely lead to modifications in the CNS and is not simply based on a mechanical instability [10].

The progress in neuroscience opens new possibilities to take a look into brain mechanisms in musculoskeletal rehabilitation. Functional MRI (fMRI) and electroencephalography (EEG) are able to provide data due to brain activity and connectivity [11, 12] in a relationship to ACL deficiency. Recently a few researchers followed these research line and started exploratory

pioneer work to add measures of brain activity in sensorimotor control after ACL injury. They detected brain areas like the frontal and parietal cortical areas related to higher executive functions, somatosensory information processing and the cerebellum due to fine-tuning in sensorimotor control which serve as a neuronal correlate to motor behavior [13–15]. It is hypothesized that the working memory as a mechanism for integration of relevant information into the movement planning may play specific a role after ACL injury [13]. Changed afferent feedback is thought to increase executive attentional control and processing activity in the somatosensory areas. These results attract new methodological approaches to look into the fronto-parietal network communication inside the brain and into brain-muscle communication by EEG functional connectivity measures [16] in ACL research. But even if the results are promising: we are still at the beginning to discover brain mechanisms after musculoskeletal injuries, but it is a start!

For future research in sensorimotor control related musculoskeletal rehabilitation this example should demonstrate the importance to open the window to the brain related to motor behavior. Only the “synchronized” look at the internal processing and the external motor output can help to discover the underlying mechanisms behind injury and damage. Neuroscience and biomechanics should work closely together in an interdisciplinary approach to reach the final goal in musculoskeletal rehabilitation research: to build a scientific base for optimized rehabilitation programs with the understanding why the programs work which will be highly beneficial for the patient.

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## PUBLICATION 8

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<b><i>Study supervision</i></b>	<b>Baumeister</b>

## ABSTRACT

After reconstruction of the anterior cruciate ligament (ACL) afferent proprioceptive information from the knee joint may be altered. In order to examine changes in central activation patterns spectral features of the EEG were measured. Patients after ACL-reconstruction and healthy controls carried out an knee-angle reproduction task in a groups x limbs x trials design. Cortical activity was recorded using international standards. FFT were conducted to determine power at Theta, Alpha-1, Alpha-2 and Beta-1. Statistics show significantly larger aberrations in the reconstructed limbs compared to the controls whereas there are no differences between the uninvolved limb controls. Brain activity demonstrates significantly higher frontal Theta power (F3, F4, F8) in both limbs of the ACL-group versus the controls and significantly higher Alpha-2 power were shown in the ACL reconstructed limb compared to controls at parietal positions (P3, P4). No such differences were found between the uninvolved side and the controls. The EEG was able to measure a change in joint position sense (JPS) at cortical level after the reconstruction of the ACL. The results of these findings might indicate differences in focussed attention with involvement of the anterior cingulate cortex (frontal Theta) and sensory processing in the parietal somatosensory cortex (Alpha-2).

**Key words:** ACL, cortical activity, proprioception, sensorimotor system, JPS



## INTRODUCTION

Ruptures of the anterior cruciate ligament (ACL) have a high incidence in athletic injuries. Resulting movement problems after ACL rupture or reconstruction have been investigated rather frequently to understand the underlying peripheral mechanisms. According to a literature review a deficient or reconstructed ACL causes biomechanical alterations (Alkjaer et al., 2002; Chmielewski et al., 2005), as well as as decreases in muscular strength (Elmqvist et al., 1988; Baumeister & Weiss, 2002) and function (Chmielewski et al., 2002; Rudolph et al., 2000). Furthermore, proprioceptive functions were found to be affected by an ACL reconstruction/deficiency (Barrett, 1991; Corrigan, 1991; Roberts et al. 2000; Bonfim et al., 2003; Katayama et al., 2004). Proprioception is a sensory modality that includes the perception of movement, force and joint position. Besides the muscle spindle there are mechanoreceptors such as Ruffini endings, Pacini corpuscles and Golgi-tendon organs in and around the human knee which are responsible for the sensation of the joint position (Gilman, 2002). A reconstruction of the ACL leads to speculations of a lack of sensory afferent information from these mechanoreceptors (Johansson et al., 1991; Wojtys & Huston, 1994; Ageberg, 2002).

There have been several studies about the joint position sense (JPS) as a proprioceptive modality in knees with a ruptured or reconstructed ACL which have demonstrated an increased error in the joint position sense paradigm in ACL patients (Barrett, 1991; Jerosch et al., 1996; Fremerey et al., 1998). However, other authors have failed to find a significant difference when comparing patients to an external control group (Roberts et al., 2000; Fisher Rasmussen et al., 2001). Different findings have often been described to methodological variety (Beynon et al., 2000). Until now it is unclear which methods of angle reproduction can detect deficits in this proprioceptive modality most efficiently.

The joint position sense and its involved receptors are the initial part of the complex sensorimotor system. The afferent information is processed in the CNS which responds to the afferent stimulus by discharging a motor (efferent) signal. This signal modulates effector muscle function by controlling the joint position sense (Riemann & Lephardt 2002). If the

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afferent information is altered after ACL-deficiency/reconstruction one can assume that the cortical information processing has changed and that such alterations might be detectable.

A few authors have measured somatosensory-evoked potentials (SEPs) to describe changes on the CNS level in ACL patients. Pitman et al. (1992) demonstrated SEPs in the cerebral cortex after direct stimulation of an intact ACL during arthroscopy in nine patients. Valeriani et al. (1999) measured SEPs and knee position sense in seven patients after ACL-lesion before and after reconstruction. They found a decreased knee position sense and a lack of P27 potential in the SEPs. Hence the authors suggested that a loss of knee mechanoreceptors can lead to modifications in the CNS. The same methodology was used by Ochi et al. (1999). They found SEPs in all ligaments which had been reconstructed as well as in all cases of a control group. On the other hand SEPs were detectable in only 15 out of 32 cases of the injured group. They concluded that a sensory re-innervation occurred in the reconstructed ACLs.

Another technique for measuring alterations at the cortical level is the ongoing electroencephalography (EEG) which was recommended as a promising method to proof the influence after ACL reconstructions on the cortical level by Kapreli and Athanasopoulos (2006). Therefore we used the EEG to detect differences in cortical activation pattern during a knee joint position task.

Most of the knee joint position studies investigate differences between groups like ACL patients and external controls. Another topic which must be taken into account is the acquisition of a new skill like a JPS task. Skill acquisition is an important feature in therapy and is defined by a increased performance (decreased error in a joint position paradigm; Schmidt & Lee 1999) and by an increased economy of used cortical resources which can also be measured by the spectral features in the ongoing EEG (Etnier et al., 1996; Smith et al., 1999; Häufner et al., 2000). So the second issue to be investigated is to look for parameters which can describe the learning process.

In summary, the aim of study was to look i) if the central information processing is influenced after an ACL-reconstruction during a sensorimotor JPS task and ii) which parameters can be used to describe the skill acquisition during therapy.

## **MATERIALS AND METHODS**

### **Subjects**

A total of 22 volunteers participated in the present study and were divided into two groups: Ten patients after ACL reconstruction (7 males / 3 females;  $27\pm 5$  years;  $76\pm 13$  kg;  $181\pm 10$  cm;  $12.5\pm 4.6$  months since reconstruction) were recruited from the University of Paderborn. Twelve healthy subjects (9 males, 3 females;  $25\pm 3$  years;  $76\pm 10$  kg;  $181\pm 8$  cm) with no history of trauma or neurological complaints of the lower limbs served as a control group. Handedness was evaluated with the EHI questionnaire (Oldfield 1971) as well as footedness based on a questionnaire asking all subjects about their preferred leg as well as which leg they use for kicking a ball or stepping on a chair. There was only one patient in the ACL group and two control subjects who were categorized as left footed according to the evaluation procedure. Both left footed subjects were also left handed. All other participants were right-footed and right-handed. Taking this evaluation in account there were five patients in the ACL group whose dominant leg was injured. Due to the statistical demands the subjects were chosen in a way that dominant legs were equally distributed in both groups (see study design; fig 1). Reconstructions were carried out by different surgeons using the quadrupled hamstring tendon autograft. All ACL patients (Lysholm Score at  $86,5\pm 3,9$ ) have been in a rehab program. Part of each rehabilitation program was a sensorimotor training. This training consisted of postural stabilisation tasks on a two-dimensional free-swinging platform, on wobbling boards, spinning tops and soft mats. The patients did not have any pain or swelling of the involved knee joint. The diagnostics showed no further problems like cartilage or meniscal damages. All subjects were recreational athletes from soccer (control: 4 / ACL: 4), swimming (3/2), handball (2/2) and fitness (3/2) and had a Tegner activity score of

five and more before injury, and a mean score of  $4.5 \pm 1.5$  at the time of the experiment. They had no history of neurological, cardiovascular or other major disorders, no current use of medications or drugs and no physical or psychological exposures (e.g. intensive training sessions, examinations) 24 hours prior to the trial. All subjects had normal or corrected to normal vision at the time of the experiment. The study was done in accordance with the rules and regulations established by the Institutional Review Board for ethical treatment of human subjects. All subjects signed an informed consent after the explanation of the testing procedure. All data were treated with confidentiality.

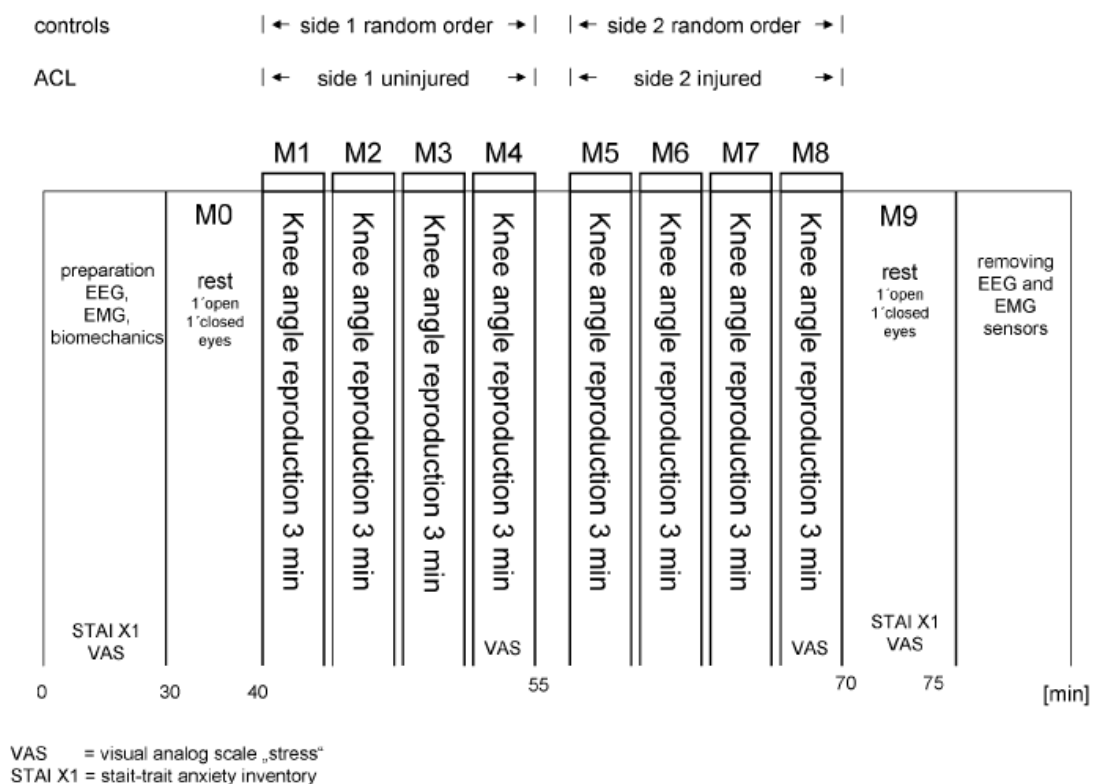


FIGURE 1. Study design

## Measures

**Performance measures.** The performance of all participants were measured in sitting position on the training machine M3 (Schnell, Germany) with an electro-goniometer

(Novotechnik, Germany) which measured the rad of lever arm motion. The results were stored on a notebook (Amilo 7400, Fujitsu-Siemens).

**Psychometric measures.** Subjective state was measured using the State Trait Anxiety Inventory (STAI) to assess anxiety at the time (state) and habitual anxiety (trait). Furthermore a visual analogue scale from 0 (no stress) to 10 (highest possible stress) measured the stress level.

**Peripheral (EMG) preparation and recordings.** Electromyographic (EMG) signals from 3 leg muscles (rectus femoris: RF; vastus medialis: VM; vastus lateralis; VL) were obtained, using disposable surface EMG-electrodes (Ag/AgCl; ARBO HealthCare, Kendall GmbH, Germany). The skin was shaved, abraded and cleaned before electrode attachment in order to reduce skin impedance and ensure good adhesion of the electrodes. The centre to centre electrode distance was 20 mm. Electrode locations were based on SENIAM guidelines (Hermens et al., 1999). The signals were preamplified with a bandwidth of 10 Hz to 500 Hz (Biovision, Germany) and transmitted to a notebook at a sampling rate of 1024 Hz.

**Central (EEG) preparation and recordings.** The EEG was recorded by a stretchable electro cap (ElectroCap Inc., USA) in accordance with standards of the international 10:20 System. During the task performance (3 min) and the rest periods in the beginning and the end (2 min) EEG was recorded continuously from 17 scalp locations (Fz, F3, F4, F7, F8, Cz, C3, C4, Pz, P3, P4, T3, T4, T5, T6, O1, O2) using the central electrode (Cz) as physical reference. All EEG data were recorded and stored using the CATEEM system (MediSyst, Linden, Germany). The signals were sampled at 512 Hz / 12 bit and amplified (DC=20 M $\Omega$ ). Before each measurement an impedance test (< 50 k $\Omega$ ) ensured a sufficient signal to noise ratio. The physiological signals were highpass filtered at 0.86 Hz. An automatic artefact detection (depending on amplitude level and signal slope) was followed by a visual inspection. Only artefact free segments were used for analysis.

**Procedures.** The study was conducted in a laboratory at the Institute of Sports Medicine (University of Paderborn). In a preliminary examination one week before the experiment, the participants were familiarized with the surroundings, the task and the procedures.

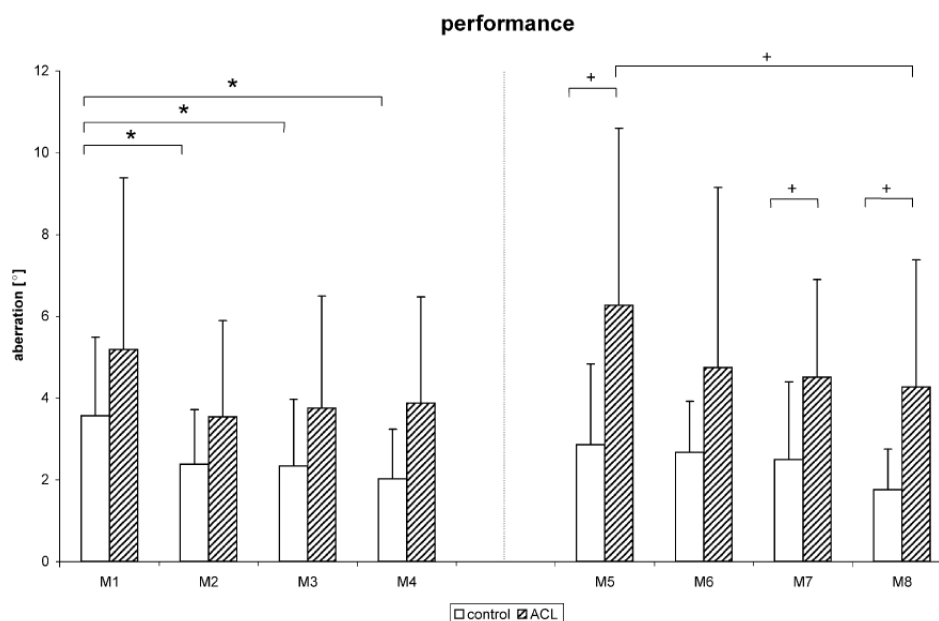
For the main part of the study electrodes for EMG (RF, VM, VL) as well as the electro cap were then attached to the subjects. They were asked to lie supine and relax for a 10 min period. After relaxing the state-trait- anxiety inventory had to be filled out. Thereafter, the subjects were seated on the training machine M3 (Schnell, Germany) with a hip flexion of 110°. The rotation axis of the dynamometer was visually aligned in relation to the lateral femoral condyle. The pad was removed from the lever arm to ensure a direct and accurate reaction of the goniometer. To avoid pain sensations the subjects wore shin pads.

After the signals were tested for quality (EMG: visually during test movements; EEG: impedance test), EEG signals were recorded for 2 min (M0; 1 min eyes open, 1 min eyes closed). Then participants were asked to reproduce a given knee angle of 40° by moving the lever arm (without any additional weight) of the dynamometer from 90° to the target angle (full extension is equivalent to 0°). A visual feedback (represented by a red horizontal line on a video monitor) was given while the target angle was to be reached as accurate as possible during five movements before each trial. After that the visual feedback was withdrawn subjects were made to repeatedly reproduce the target angle in a 3 min trial at their individual pace. Every participant had to perform eight trials, four with each side (M1-M4 / M5-M8; fig.1). The control subjects started with one extremity chosen by random whereas the ACL patients always performed with their uninvolved side first. (fig.1). The subjects were instructed to reproduce the target angle as accurate as possible and every time when they reached the target position they were advised to stay there for three seconds. During the trials without visual feedback (M1-M8) knee angle, EMG and EEG were recorded continuously.

**Signal processing.** The error in reproduction accuracy was calculated as the aberration between the target angle with vs. without visual feedback. Errors were averaged over each trial.

The raw EMG signals from the RF, VM and VL were full-wave rectified and integrated during each trial for 1000 ms starting when the subjects had reached the target angle (IEMG<sub>1000</sub>; DasyLab 7.0 software package). The whole movement activity was defined as [RF IEMG<sub>1000</sub> + VM IEMG<sub>1000</sub> + VL IEMG<sub>1000</sub>] during each trial (M1-M8). Afterwards each muscle activity was related to the whole movement and percentages (%) were calculated to detect changing strategies in neuromuscular activation.

In the EEG signals, Fast Fourier Transforms (FFT) were calculated on 50% overlapped, 512 sample Hanning windows for all artefact free segments with the CATEEM system (MediSyst, Linden, Germany). The power spectra were divided into different frequencies: Theta (4.75 – 6.75 Hz), Alpha 1 (7.0 – 9.5 Hz), Alpha 2 (9.75-12.5 Hz) and Beta 1 (12.75 – 18.5 Hz). Average power spectra were computed across all trials for performance (M1-M8) in each subject.



**FIGURE 2.** Performance during the JPS task (M1-M8) in ACL patients and controls. (significance level: \*/+ p<0.05)

### Statistical Analysis.

For statistical analysis SPSS 12.0G software was used. All results are given as mean values and standard deviation. The Kolmogorov-Smirnov (K-S) test was used to determine if

variables fitted the Gaussian distribution. To examine differences in the calculated parameters a General Linear Model (GLM; MANOVA) for repeated measures was conducted in a two (ACL vs. control) x two (extremities) x four (measurement periods) design. Compound symmetry, or sphericity, was verified by the Mauchley test. When the assumption of sphericity was not met, the significance of F-ratios was adjusted according to the Greenhouse-Geisser procedure. T-tests were used post hoc to detect significant differences between and within groups. Otherwise, non parametric tests were used to analyse differences between ACL patients and controls (Mann Whitney-U test) or M1-M8 (Friedman/Wilcoxon-test). The outcome of statistical calculations were declared significant if  $p \leq 0.05$ .

## RESULTS

**Task performance.** The ACL patients reproduced the target angle with a significantly larger error compared to the controls (fig.2; tab.1). GLM detected an interaction effect with practice in the first part of the experiment ( $F_{3,60}=3.082$ ;  $p=.036$ ). Post hoc tests demonstrated these practice effects in the control group between M1-M2 ( $T(2.653)$ ;  $p=.022$ ), M1-M3 ( $T(2.482)$ ;  $p=.030$ ) and M1-M4 ( $T(2.948)$ ;  $p=.012$ ). In the ACL patients there was an effect within the group only between M5-M8 ( $Z(-2.293)$ ;  $p=.022$ ).

**Psychometric Measures.** The State-Trait Anxiety (STAI) scores showed no differences between and within groups for all the parameters. The subjective stress level (VAS) started off low and then increased in both groups with highest scores after M8 (control) and at the end (ACL). However there was no significance between or within groups.

**EMG Parameters.** The involved VL activity related to the whole movement [%] showed significant increases in the ACL group compared to the controls at M5 and M6 (Fig. 4; tab.1). The differences in the VM activity related to the whole movement showed decreased activity in the ACL patients in the second part of the experiment ( $F_{1,20}=3.688$ ;  $p=.069$ ), but the



findings did not reach significance (fig.3). No practice effects could be detected between M1-M4 and M5-M8 in all calculated muscles.

Table 1: Significance level between ACL patients and controls in performance, EMG and EEG parameters during the JPS task (M1-M8)

Parameter	M1		M2		M3		M4		M5		M6		M7		M8	
	Z	P	Z	P	Z	P	Z	P	Z	P	Z	P	Z	P	Z	P
Abs. error [°]									-2.440	.015			-2.308	.021	-2.506	.012
EMG VL [%]									-2.176	.030	-2.308	.021				
Theta F3	-2.642	.008	-2.678	.007	-2.180	.030	-2.343	.019	-2.673	.008	-2.279	.023	-1.947	.050	-2.606	.009
Theta F4	-2.704	.007	-2.835	.005	-2.607	.009	-3.004	.019	-2.939	.003	-2.408	.016	-2.377	.017	-3.038	.002
Theta F8	-2.345	.017	-2.845	.003	-2.215	.025	-2.869	.003	-2.410	.016	-2.638	.008	-2.177	.029	-2.573	.010
Alpha-2 P3													-2.012	.044	-2.608	.009
Alpha-2 P4									-2.210	.027			-2.243	.025	-2.642	.008

ACL, anterior cruciate ligament; EEG, electroencephalography; EMG, Electromyographic; JPS, joint position sense.

**EEG Parameters.** A comparison of all EEG parameters in the resting state (M0) between both groups showed no differences. In the control group the EEG frequency bands remained equal between both sides.

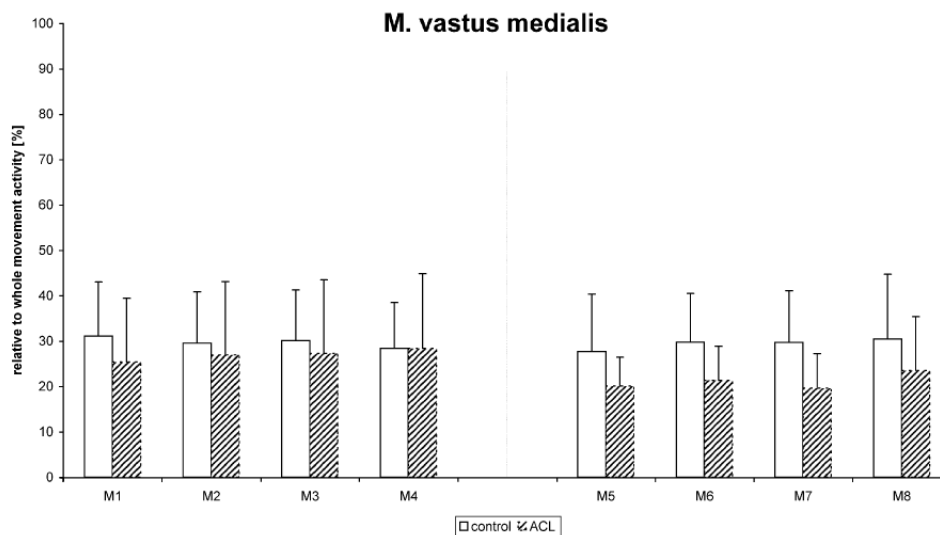


FIGURE 3: VL EMG activity related to whole movement activity (%) during the JPS task (M1-M8) in ACL patients and controls. (significance level: \*  $p < 0.05$ )

But EEG power spectra demonstrated differences between ACL patients and controls in the Theta and “fast” Alpha (Alpha 2) frequency bands. Statistical analysis revealed that the ACL patients exhibited significantly more power at frontal **Theta** than controls during the joint

position reproduction at F3 (*tab.1, fig.2*), F4 (*tab.1, fig.3*) and F8 (*tab.1*). Furthermore, the statistical analysis revealed increased Theta power with practice as a interaction effect in the first (Fz:  $F_{3,60}=3.196$ ;  $p=.030$ ; post hoc controls M2-M3 and M1-M4) and in the second part of the experiment (M5-M8; F3:  $\chi^2=7.867$ ;  $p=.049$ ; post hoc controls M5-M7).

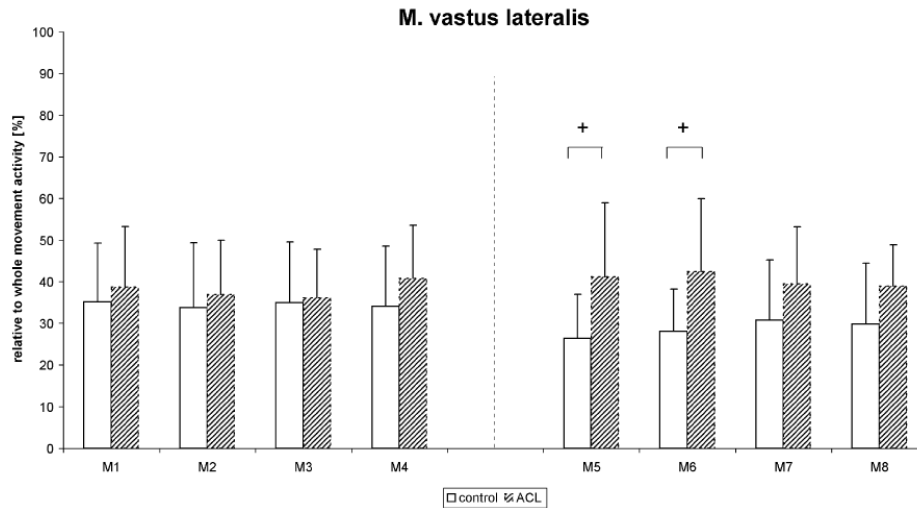


FIGURE 4: VM EMG activity related to whole movement activity (%) the JPS task (M1-M8) in ACL patients and controls.

Additionally, the controls had a significantly greater **Alpha-2** power in parietal areas at P3 (*tab.1, fig.5*) and P4 (*tab.1, fig.4*) compared to the ACL group. Within the groups there were increased Alpha-2 power with practice in the first part at T4 [ $F_{3,60}=4.579$ ;  $p=.006$ ; post hoc ACL: M1-M2 ( $T(-2.928)$ ;  $p=.017$ ), M1-M3 ( $T(-2.415)$ ;  $p=.039$ )] and in the second part of the experiment at P3 (*fig.5*;  $\chi^2=14.958$ ;  $p=.002$ ; post hoc control: M5-M6 ( $Z(-2.049)$ ;  $p=.040$ ; M5-M8 ( $Z(-2.002)$ ;  $p=.045$ ; post hoc ACL: M5-M8 ( $Z(-2.552)$ ;  $p=.011$ ) and T4 [ $F_{3,60}=3.424$ ;  $p=.023$ ; post hoc control: M5-M6 ( $T(-2.209)$ ;  $p=.049$ ); M6-M7 ( $T(-2.808)$ ;  $p=.017$ ); M5-M7 ( $T(-4.360)$ ;  $p=.001$ ]).

The **Alpha-1** frequency band showed no effects between both groups, but an increasing power with practice only between M1 and M4 widespread over the cortex in detail at F3 ( $F_{3,60}=3.843$ ;  $p=.014$ ), F4 ( $F_{3,60}=4.442$ ;  $p=.007$ ), F7 ( $F_{2,188,60}=3.385$ ;  $p=.024$ ), F8 ( $F_{3,60}=5.528$ ;  $p=.002$ ), Cz ( $F_{3,60}=3.871$ ;  $p=.013$ ), P3 ( $F_{3,60}=5.776$ ;  $p=.015$ ), T4 ( $F_{3,60}=5.150$ ;  $p=.003$ ), T5 ( $F_{2,411,60}=4.840$ ;  $p=.004$ ), T6 ( $F_{3,60}=4.844$ ;  $p=.004$ ), O1 ( $F_{2,565,60}=7.367$ ;  $p=.001$ ) and O2

( $F_{3,60}=3.390$ ;  $p=.049$ ). The post hoc analysis revealed this effect in both groups at the mentioned positions. There were no between or within effects in the **Beta-1** spectral power.

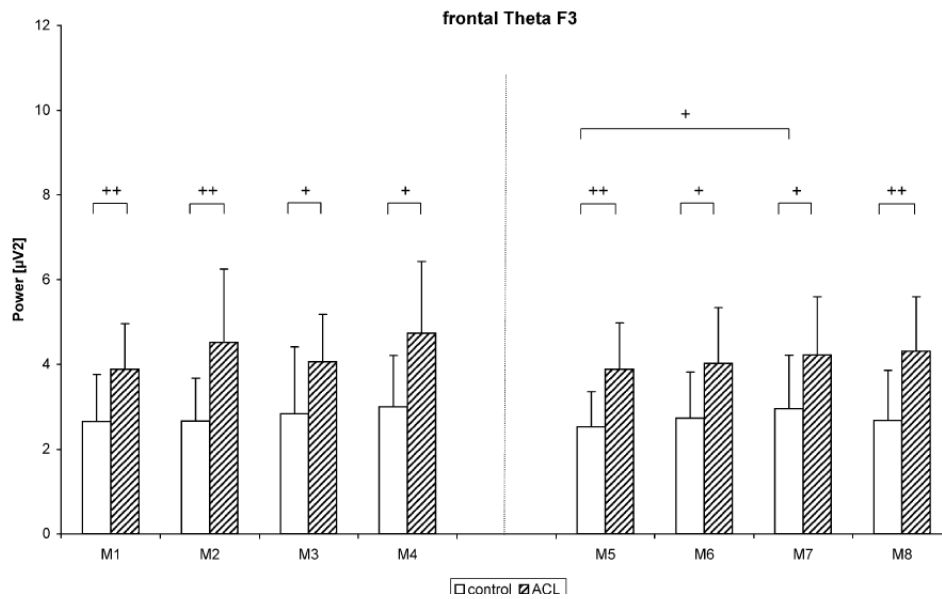


FIGURE 5: Left-frontal Theta power at electrode position F3 during the JPS task (M1-M8) in ACL patients and controls. (significance level: +  $p<0.05$ ; ++  $p<0.01$ )

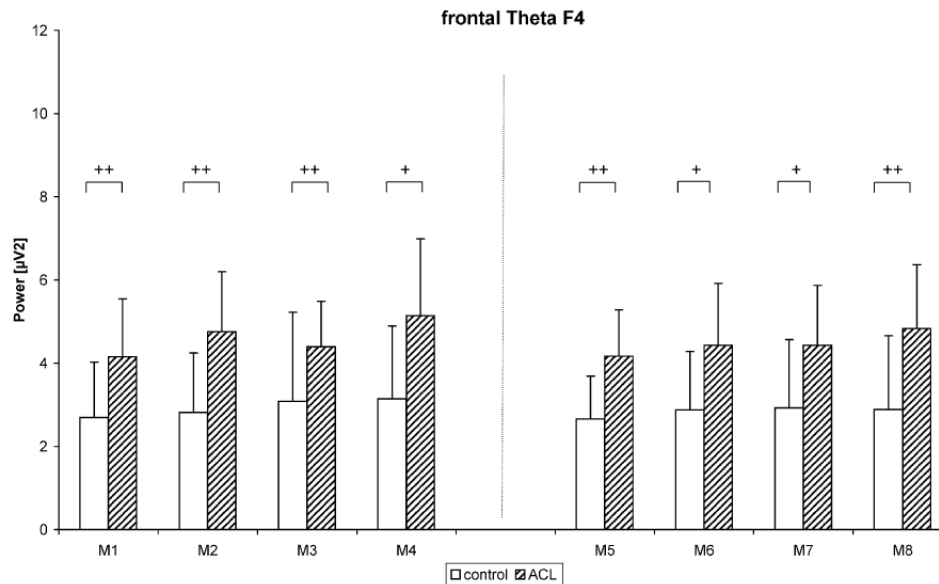


FIGURE 6: Right-frontal Theta power at electrode position F4 during the JPS task (M1-M8) in ACL patients and controls. (significance level: +  $p<0.05$ ; ++  $p<0.01$ )

## DISCUSSION

The integration of proprioceptive afferent information is an important issue of motor control particularly after ACL reconstructions. Different authors speculate about changes in brain activity after ACL reconstruction (Johansson 1991; Wojtys & Huston 1994; Ageberg 2002; Sjölander et al. 2002). However, the goal of the present study was to investigate if cortical activities that occur during the conduction of a JPS task were altered after the reconstruction of the ACL. The term *after reconstruction* covers at least three factors which might be represent important differences compared to a healthy control group: the injury, the reconstruction and the rehabilitation program. Each of them or a combination could be the cause for the measured differences. Therefore, we will address all factors together as *after reconstruction* throughout the discussion. Furthermore skill acquisition was accomplished by characterizing how spectral features of the EEG change as subjects learn to perform a task more efficiently.

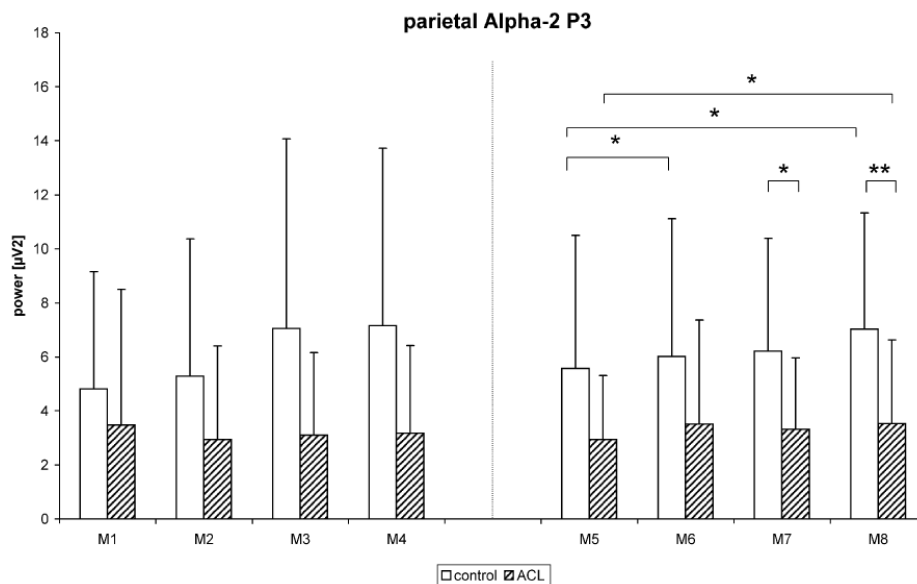


FIGURE 7: Left-parietal Alpha-2 power at electrode position P3 during the JPS task (M1-M8) in ACL patients and controls. (significance level: \*  $p < 0.05$ ; \*\*  $p < 0.01$ )

### Influence of ACL reconstruction

The involved leg of the ACL group reproduced the target angle with a significantly larger error compared to the control group whereas the statistical analysis could not detect any significance between the controls and the uninvolved extremity of the ACL group. This result is supported by different authors.

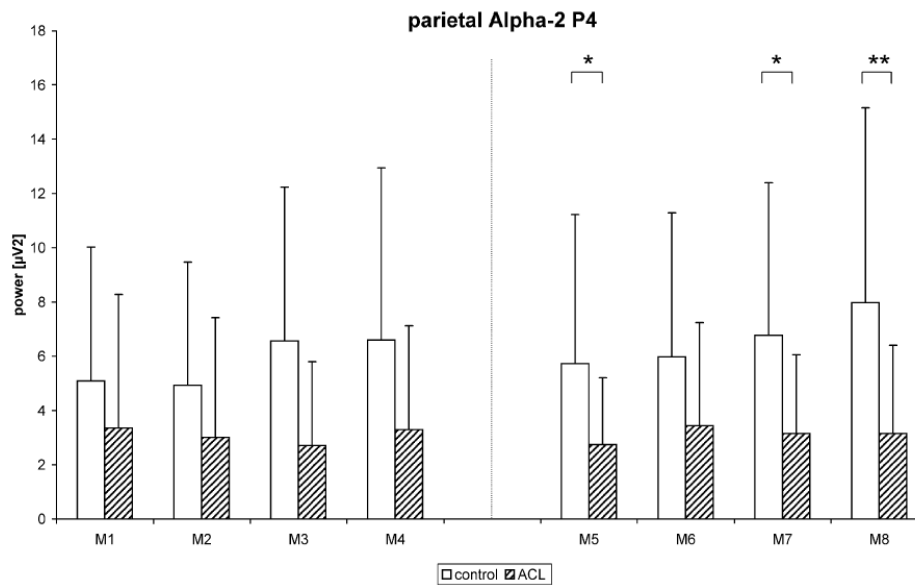


FIGURE 8: Right-parietal Alpha-2 power at electrode position P4 during the JPS task (M1-M8) in ACL patients and controls. (significance level: \*  $p < 0.05$ ; \*\*  $p < 0.01$ )

They found a significant increase in reproduction error as an effect after ACL reconstruction or deficiency (Barrett, 1991; Jerosch et al., 1996). These authors argue that altered afferent information has taken place due to damage of different receptor structures in the ACL. The main types of mechanoreceptors, Ruffini-endings, Pacinian corpuscles and Golgi-tendon organs contribute to the joint position sense (Gilman, 2002). It was suggested that JPS tasks might be able to detect deficits in proprioception. In contrast to that opinion, other authors have failed to demonstrate such differences (Roberts et al., 2000; Fisher-Rasmussen et al., 2001). They discuss the idea that muscle-spindles and Golgi-tendon organs could be responsible for JPS over most of the working range. Furthermore most of the studies are not comparable because of serious differences in the utilized methodology like active or passive reproduction, body position during the testing, time since reconstruction and if the contra

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lateral side or an external group served as control condition. Thus it could be questioned if the used JPS testing procedures relying on a single biomechanical parameter (error) can detect alterations in the complex sensorimotor system.

Neuromuscular strategies during the given task were part of the sensorimotor system. The ACL patients showed a significant increase in participation of the vastus lateralis (VL) while performing with the reconstructed side compared to the control group. This leads to speculations of an altered neuromuscular strategy in ACL patients as a reaction to their decreased participation of the vastus medialis (VM) muscle related to the whole movement activity. In open chain isometric and isokinetic measurements the vastus medialis often demonstrates a decreased activity due to ACL damages (Freiwald et al., 1999; Fink et al. 1994). This finding may be explained as having a reflex origin (St Clair Gibson, 2004), but this change in activation strategy during the goal-directed task may require central modifications as well.

This leads to the most important finding of this study: the influence after ACL reconstruction at the biomechanical and neuromuscular level was associated with significant changes in prominent spectral features of the ongoing EEG: the frontal Theta and the parietal Alpha-2 power.

The ACL patients demonstrated significantly higher frontal Theta Power (F3, F4, F8) from M1 to M8 compared to the healthy controls. This finding was supported by numerous cognitive and visuomotor studies which describe frontal Theta power as an indicator of focussed attention and task complexity. The frontal Theta frequency is thought to be generated in the anterior cingulate cortex (ACC; Bordmann area 24 and 32; Gevins et al., 1997). The ACC source for the attention related frontal Theta is consistent with the results from other neuroimaging studies (LaBerge et al., 1990; Vogt et al., 1992), a monkey model (Tsujimoto et al., 2006) and brain lesion studies (Janer & Pardo, 1990) that this anatomical region is an important part of the human attentional system. All neuroimaging studies show that frontal Theta is enhanced, relative to a resting situation, when subjects engage in complex, attention demanding tasks.

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In the current study, we found that the ACL patients performed with increased frontal Theta power compared to the healthy control group which might reflect a higher state of focussed attention during the performance after ACL reconstruction. Furthermore the situation may appear more complex to the ACL group which may require a higher attention level. Gevins et al. (1997) showed an increase in frontal Theta with higher complexity of the tasks during a cognitive task. The same relation of frontal Theta power to complexity was described by Smith et al. (1999) at the fronto-midline scalp position. Their subjects had to play a video game ("space fortress") in a partial as well as in a more complex task. Slobounov et al. (2001) confirm this result during a tracking task (video game "frustrated maze") with and without time pressure.

A second important finding in association with frontal Theta power and focussed attention is the significant higher Theta power in ACL patients even on their uninvolved side. That means the ACL reconstruction also influences the uninvolved side. This was shown by Jerosch et al. (1996) who investigated the influence of ACL-deficiency in a JPS task and found significantly more errors on both the involved and uninvolved side. The current findings support the idea of the involvement of the "healthy" leg after ACL reconstruction. Alternatively, this finding could be caused by the rehabilitation program. One can speculate about the need of "cortical attention" which is necessary during the rehabilitation program to re-learn motor programs. Further studies should focus on that topic in more detail. However, the idea of using the uninvolved side as a reference during therapy should be seriously reconsidered.

The second prominent frequency band which shows significant differences between both groups is the Alpha-2 in parietal areas of the cortex. Alpha is the dominant frequency in the human scalp of the EEG in adults and is thought to be generated in widespread areas of the cortex due to cortico-cortical and thalamo-cortical interactions (Niedermeyer & Lopes da Silva 2005). Alpha activity is often described as a form of cortical idling with its amplitude inversely related to the number of neuronal populations activated during cognitive and motor processes (Gevins et al. 1997; Niedermeyer & Lopes da Silva 2005). Usually the Alpha frequency is divided into two subgroups: a "slow" (Alpha-1, 7.0-9.5 Hz) and a "fast" (Alpha-2,

9.75-12.5 Hz) component. Whereas the slow alpha band component (Alpha-1) is attenuated over broad regions of the cortex and is thought to be a non-specific attentional and expectancy process indicator, the fast Alpha (Alpha-2) reflects task-specific demands in parieto-occipital regions in the area of the somatosensory cortex. The significant lower Alpha 2 parietal (P3, P4) power at the involved side of the ACL group indicates the notion of higher cortical activation in the parietal regions compared to the controls. The ACL patients have to deal with three new factors, the JPS task, the altered muscle coordination and the altered afferent information from the knee joint, whereas the situation involves only the first factor for the controls. Therefore, the ACL patients potentially require more neuronal resources in the parietal cortex while performing the more complex situation with their reconstructed side. Similar results are described for visuomotor tasks. In accordance with this study Häufler et al (2000) found increased Alpha power in marksmen compared to novice shooters. The results indicate that there is a relative economy in the cortical process of marksmen depending on their skill level characterized by lower neuronal activity demonstrated as higher Alpha-2 power.

Is there a conjunction between the frontal Theta power and the parietal Alpha-2 component? Both parameters were consistently described in cognitive and visuomotor task as indicators of task complexity in conjunction with the load of the working memory (Gevins et al., 1997; Smith et al., 1999; Slobounov et al., 2001). The general concept of working memory suggests that a limited capacity system which temporarily maintains and stores information, supports human thought processes by providing an interface between perception, long-term memory and action (Baddeley, 2003). This theoretical framework differentiates central executive functions from storage functions related to the two subsystems: the phonological and the visuo-spatial loop. In that picture, the central executive (CE) is assumed to be an attentional control system responsible for strategy selection, control and coordination of various processing tasks. The anatomical location of the central executive processing were traditionally related to the frontal lobe (for a review see Collette & van der Linden, 2002). Recent studies demonstrate growing evidence, that this central executive functions rely on a



fronto-parietal network. Such associations were found with different methods, functional Magnet-Resonance-Imaging (Kondo 2004; Osaka 2004), Positron-Emission-Tomography (Collette 1999) and EEG (McEvoy et al., 2001; Sauseng et al, 2004). Sauseng et al. (2005) investigated the coherent EEG activity within a fronto-parietal network by various demands of executive functions. They found an increase of long-range coherence reflecting the fronto-parietal network. They concluded that fronto-parietal EEG coherence in Theta and Alpha-2 reflects central executive functions of the working memory.

With this theoretical background we suggest a novel model of the brain as an executive control organ which plays a major role even in peripheral injuries and the corresponding rehabilitation program. Based on the literature the increased frontal Theta power and decreased parietal Alpha-2 power related to the reconstruction of the ACL may demonstrate a higher load of the CE functions of the working memory. It can be speculated that strategy selection, control and coordination in this JPS task need more neuronal resources compared to a healthy control group.

In summary, for the first time this study demonstrates that cortical activity is altered after the reconstruction of the ACL. The frontal Theta and the parietal Alpha-2 power seem to be indicators of this effect. They may be parts of a fronto-parietal network reflecting the central executive functions of the working memory. In conjunction with the performance in JPS and neuromuscular activity, the EEG is applicable for monitoring changes after ACL reconstruction.

### **Influence of skill acquisition**

The goal of the second part of the study was to investigate EEG correlates of neural adaptation that happen during the acquisition of the joint position sense task. EEG parameter changes were examined while participants learned how to perform the JPS task more efficiently.

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The behavioural data indicate that practice indeed improved task performance. In particular, this occurred in the first part of the experiment with decreased size of errors in the control group with ongoing task practice. In the second part of the experiment, practice increased the accuracy in the ACL-group characterized by a decreased size of error. According to Schmidt & Lee (2000) the decrease of the error with practice demonstrates skill acquisition. The behavioural data were associated with changes in different prominent spectral features of the ongoing EEG.

EEG activity in the Alpha frequency band increased with practice in both groups. Practice-related changes in Alpha-power might be interpreted as indicating that progressively fewer cortical neurons were activated during task performance (Niedermeyer & Lopes da Silva, 2005). Häufler et al. (2000) described this as a strategy to use neuronal resources more efficiently. As mentioned above the Alpha frequency is often divided into two different components: the Alpha-1 ('slow') frequency shows increased power over widespread areas of the cortex and represents non-specific attention and expectancy processes (Smith et al., 1999). Both groups showed increases in Alpha-1 power in the JPS task, but only in the first part of the experiment. One can speculate about that as a familiarization process. Participants of both groups needed to find the right strategies for performing well. This phase was described by Schmidt & Lee (1999) as the cognition phase in skill acquisition. In the second part, subjects were already familiar with the task and the surroundings, so that there was no further increase in Alpha-1 power.

In contrast the higher Alpha frequency component ('fast' Alpha-2) tended to be attenuated in the posterior areas (PTO = parietal-temporal-occipital area) of the scalp. An increase in power, especially in the parietal regions, according to practice-related increase in accuracy might be interpreted as a strategy of increasing economy. Following the notion that the alpha magnitude is inversely related to the proportion of neurons activated during the task, this economy consist in a more effective use of neuronal resources during skill acquisition. If the parietal Alpha-2 power is part of a fronto-parietal network representing the central executive function of the working memory it can be assumed that an increased power is an indicator of

a decrease in working memory load. The results of this study demonstrate that this power increase happened in both groups independent from differences between ACL patients and a healthy control group. Therefore, the findings in the Alpha frequency seemed to demonstrate that it is consistent with principles of human motor learning even after the reconstruction of the ACL: efficiency and speciality of adaptation.

## **PERSPECTIVES**

For the first time we have measured cortical activation patterns during a sensorimotor JPS task and were able to detect the influence of an ACL injury and reconstruction at the cortical level. With the theoretical framework of a working memory it seemed to be possible to develop a model for ACL patients. Thus, in follow up studies sensorimotor tasks should be designed to control and manipulate the working memory load to accept or reject the model. With the possibility to measure cortical alterations after ACL reconstruction, training/therapy could be evaluated and it may be possible to investigate if the injury, the reconstruction, the rehabilitation or a combination is responsible for the changes demonstrated in this study. The effectivity of exercises especially in the field of sensorimotor tasks could be controlled related to cortical activation. In this case the other proprioceptive modalities like the sense of movement and the sense of force should be investigated to receive a detailed central processing model of deficits after ACL reconstruction due to different proprioceptive information.

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## PUBLICATION 9

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## ABSTRACT

Afferent proprioceptive information from the knee joint may be altered due to a reconstruction of the anterior cruciate ligament (ACL) which could result in changes of cortical activity. The aim of the study is to look if force sensation and cortical activation measured by EEG are influenced by an ACL-reconstruction when performing a force reproduction task.

Nine patients after ACL reconstruction and nine healthy controls were asked to reproduce 50% of their maximal voluntary isometric contraction (MVIC) reproduction. EEG power values related to frequency bands and the error in reproduction were collected while performing the force reproduction. The aberration error demonstrated no significant differences between groups. The cortical activity results in significant higher frontal Theta power during the force reproduction task with the reconstructed limb (F3 and Fz:  $p < .05$ ) of the ACL group compared to the controls.

The EEG was able to measure changes in electrocortical activity after ACL-reconstruction in force reproduction, whereas performance data was not affected. The results were discussed in terms of differences in attentional control with involvement of the anterior cingulate cortex related to higher frontal Theta power in the ACL patients.

**Key Terms:** ACL, cortical activity, EEG, proprioception, force control

## INTRODUCTION

The anterior cruciate ligament (ACL) is an important mechanical structure which maintains knee joint integrity. But knee joint function is complex and does not depend on mechanical stability alone. It is also based on sensorimotor function, a dynamic interaction between the central nervous and musculoskeletal system including proprioceptive structures.<sup>1</sup> Peripheral proprioceptive information plays an important role within the sensorimotor system and after knee injuries. Proprioception is divided in three different modalities: (1) the sensation of motion, (2) the sensation of joint position and (3) the sensation of force or tension, which is commonly assessed by force reproduction and is important for goal directed movements and joint stability.<sup>2</sup> Muscle spindles and mechanoreceptors such as Ruffini endings, Pacini corpuscles and Golgi tendon organs are responsible for these sensations. An ACL injury leads to a disturbance of the sensorimotor system due to a loss or damage of the mechanoreceptors causing changes in motor behavior.<sup>3,4</sup> Therefore an ACL rupture often leads to chronic knee instability<sup>4</sup> as a consequence of a weak quadriceps and a deficit in sensorimotor function.<sup>5</sup>

With regards to impaired sensorimotor function after ACL injury/reconstruction studies have demonstrated deficits in motor behavior using sensorimotor tasks which are closely related to sensation of motion<sup>6,7</sup> and joint position<sup>8-10</sup> In contrast, the perception of force has been largely ignored in investigations of sensorimotor function related to ACL injuries. To our knowledge there is only one study which evaluates the modality of force sensation. Heroux et al.<sup>11</sup> examined the ability of weight discrimination after ACL injury compared to healthy controls. The results demonstrated a reduced acuity of the involved leg to detect differences in weight after ACL-injury compared to the uninvolved. Even if the functional sensorimotor deficit seems to be well evaluated the underlying mechanisms have not been clear.

It has been suggested that the sensorimotor dysfunction relies on changes in the CNS. In a recent study Kapreli et al.<sup>12</sup> investigated changes in brain activation pattern in ACL injured patients compared to controls during knee extension/flexion movements using functional MRI technology. They were able to demonstrate that ACL deficiency can cause reorganization in



several sensorimotor cortical areas. The first study which related motor control and CNS processing after ACL reconstruction was conducted by Baumeister et al.<sup>10</sup> They demonstrated an increased error in performance and changes in EMG activity in ACL patients. At the cortical level the patients show an increase of frontal Theta power values and decreases in parietal Alpha-2 power. The results were discussed with modifications of the working memory with frontal Theta related to a higher focussed attention and parietal Alpha-2 to differences in sensory information processing in the somatosensory cortex.

All studies demonstrate that an ACL injury may be far more than a simple peripheral musculoskeletal impairment but demonstrate cortical changes. Therefore it is worth to investigate electrocortical parameters in order to provide a clinical insight into the functional alterations of brain activity after an ACL reconstruction which might have an important impact on the design of rehabilitation programs.

Thus, the purpose of the study is to investigate the effect of an ACL reconstruction on motor behavior and cortical activity by investigating motor performance and brain activation in a force matching task. On the basis of previous literature investigating this topic, it was hypothesized that there would be a decrease in precision of force reproduction and a proof of electrocortical changes with a special focus on frontal Theta and parietal Alpha-2 power in the ACL reconstructed patients compared to their healthy counterparts.

## **MATERIALS AND METHODS**

### **Subjects**

A total of 18 volunteers participated in the present study and were divided into two groups: Nine patients after ACL reconstruction and nine controls. The ACL patients (7 males / 2 females;  $25 \pm 5$  years;  $76.8 \pm 12.2$  kg;  $182 \pm 10$ ) were recruited from the University of Paderborn. Due to the homogeneity of the group an isolated ACL injury (no further cartilage or meniscal damages) in the right dominant extremity was present in all patients. Footedness was based on a questionnaire asking about the participant's preferred leg as well as which leg they use

for kicking a ball or stepping on a chair.<sup>10</sup> The surgery for ligament reconstruction had been  $12.0 \pm 4.7$  months before the time of the study. Reconstructions were carried out by three different surgeons using the quadrupled hamstring tendon autograft. All ACL patients have been in a rehab program and must not have any pain or swelling of the involved knee joint and followed a rehab program under the supervision of physical therapists. All patients had a Tegner activity score<sup>13</sup> of five and more before injury, and a mean score of  $4.8 \pm 2.0$  at the time of the experiment and were recreational athletes from soccer ( $n=6$ ) and handball ( $n=3$ ).

Nine healthy subjects (7 males, 2 females;  $24 \pm 3$  years;  $73.0 \pm 10.2$  kg;  $181 \pm 9$ ) with no history of trauma of the lower limbs were matched by leg dominance, age and sport participation and served as a control group.

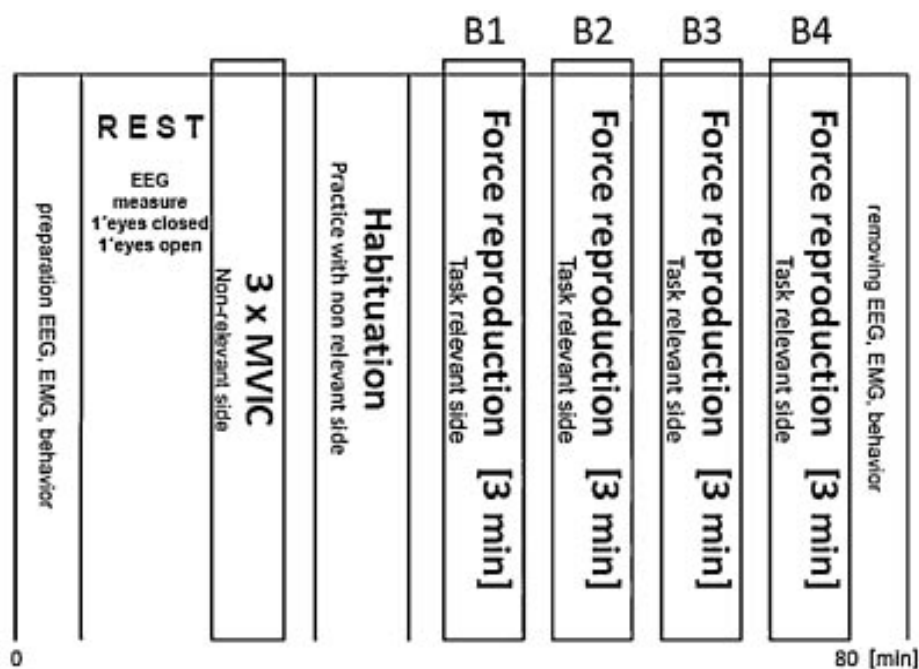


FIGURE 1. Study design

(non relevant side = uninjured/ACL and matched/control; injured side = involved/ACL and matched/control)

All participants had no history of neurological, cardiovascular or other major disorders, no current use of medications or drugs and no physical or psychological exposures (e.g. intensive training sessions, examinations) 24 hours prior to the trial. The study was done in

accordance with the rules and regulations established by the Institutional Review Board for ethical treatment of human subjects. All subjects signed an informed consent after the explanation of the testing procedure. All data were treated with confidentiality.

### **Experimental procedures**

In a preliminary examination the participants were familiarized with surroundings, the task and the procedures one week before the experiment.

For the main part of the study electrodes for EMG (RF=M. rectus femoris, VM=M. vastus medialis, VL=M. vastus lateralis) as well as the EEG electro cap were attached to the participants. All subjects were seated on the training machine M3 (Schnell, Germany) with a hip flexion of 110° and the knee flexion fixed at 90°. The rotation axis of the dynamometer was visually aligned in relation to the lateral femoral condyle.<sup>10</sup> Thereafter the signals were tested for quality (EMG: visually during test movements; EEG: visually and impedance test), an EEG measurement was conducted at rest for two minutes (1' eyes open and 1' eyes closed).

During three maximal voluntary isometric contractions (MVIC=100% reference) of the quadriceps isometric torque was obtained from the non-relevant extremity which is not used in the force reproduction testing. Each subject was required to fold the arms across the chest and was given verbal encouragement to achieve a maximal voluntary effort level.

After a habituation period (3min at their own pace) where the participants perform force reproductions with and without visual feedback with their non-relevant extremity subjects were asked to complete four testing blocks (B1-B4; Fig. 1) with their involved (ACL) or matched extremity (controls). Each block consisted of a practice period and a reproduction period. The practice period consisted of subjects performing 5 isometric knee extensions to 50% of their MVIC. Visual feedback was provided in the form of a red horizontal line on a video monitor which denoted the target force. Participants were instructed to reach the target force as accurately as possible. For the reproduction period subjects were required to perform the same task as described above without visual feedback and were instructed to

repeatedly reproduce the target force (50% MVIC) for 3 min at their individual pace. During the trials without visual feedback (B1-B4) isometric torque, EMG and EEG were recorded continuously.

## Measures

**Motor behavior** . The reproduction performance of all participants was quantified in sitting position on the training machine M3 (Schnell, Germany). Isometric forces were measured and the results were stored.

**Peripheral (EMG) preparation and recordings.** Electromyographic (EMG) signals from of the M. rectus femoris (RF), M. vastus medialis (VM) and M. vastus lateralis; (VL) were obtained, using disposable surface EMG-electrodes (Ag/AgCl; ARBO HealthCare, Kendall GmbH, Germany). Prior to electrode placement the skin was shaved, cleaned and abraded before electrode attachment in order to reduce skin impedance and ensure good adhesion of the electrodes. The centre to centre electrode distance was 20 mm. Electrode locations were based on SENIAM guidelines. The signals were pre-amplified with a bandwidth of 10 Hz to 500 Hz (Biovision, Germany) and transmitted to a notebook at a sampling rate of 1024 Hz.

**Central (EEG) preparation and recordings.** The EEG was recorded by a stretchable electro cap (ElectroCap Inc. USA) in accordance with standards of the international 10:20 system. EEG data was collected continuously from 17 scalp locations (Fz, F3, F4, F7, F8, Cz, C3, C4, Pz, P3, P4, T3, T4, T5, T6, O1, O2) using a common average reference montage.<sup>14</sup> The signals were sampled at 512 Hz / 12 bit and amplified (DC=20 M $\Omega$ ). Before each measurement an impedance test ensured a sufficient signal to noise ratio. The physiological signals were high pass filtered at 0.86 Hz. Only artifact-free segments were used for analysis (for a detailed reference based description see <sup>10</sup>).

**Signal processing.** The error in reproduction accuracy was calculated in the aberration between the target force with (practice period) and without visual feedback and was averaged in each block.

The raw EMG signals from the RF, VM and VL were full-wave rectified and integrated for each trial for 1000 ms starting when the subjects have reached the target angle (IEMG<sub>1000</sub>; DasyLab 7.0 software package). The whole movement activity was defined as [RF IEMG<sub>1000</sub> + VM IEMG<sub>1000</sub> + VL IEMG<sub>1000</sub>] during each trial (B1-B4). Afterwards each muscle activity was related to the whole movement and the percentage (%) was calculated to detect changing strategies in neuromuscular activation.

In the EEG signals, Fast Fourier Transforms (FFT) was calculated with 50% overlapped, 512 sample Hanning windows for all artifact free segments. Mean absolute EEG spectral power was calculated for each block and divided into different frequencies: Theta (4.75 – 6.75 Hz), Alpha 1 (7 – 9.5 Hz), Alpha 2 (9.75-12.5 Hz) and Beta 1 (12.75 – 18.5 Hz) Beta 2 (18.75 – 35 Hz). For the statistical analysis, a logarithmic (log) transformation of the power values was used to stabilize variances across subjects. The transformed data in the different frequency bands were computed across all blocks (B1-B4) for all single electrode positions in each subject.

#### Statistical Analysis

**Sample size calculation.** Adequate sample size was determined using means and standard deviations of frontal Theta power reported previously.<sup>10</sup> This gave a required n of 9 for each group (using a two-tailed  $\alpha=0.05$ , a  $\beta=0.2$  (or power of 80%). Based on frontal Theta power a total of 9 subjects in the ACL and in the control group were, therefore, deemed appropriate to provide adequate statistical power.

**Data analysis.** For statistical analysis SPSS 14.0G software was used. All results are given as mean values and standard deviation. Due to the small sample size the Shapiro-Wilks test was used to ensure that all variables fit the Gaussian distribution. Subject characteristics of both groups were compared using independent t-tests. To examine cortical activity in different frequencies an ANOVA with repeated measures (between-subject factor: group x within-subject factor: block) was conducted. Compound symmetry, or sphericity, was verified by the Mauchley test. When the assumption of sphericity was not met, the significance of F-

ratios was adjusted according to the Greenhouse-Geisser procedure. Independent t-tests were used post hoc to detect significance between groups. If a significant main effect of block appeared, paired t-tests were made. The outcome of statistical calculations were declared significant if  $p \leq 0.05$ . In the event of a significant main effect of block we used Bonferroni adjustment to assess pairwise comparisons of the estimated means to minimize the likelihood of a type I error. In case of significances effect sizes were calculated according to Hedges'  $d$ , a bias corrected modification of Cohen's  $d$ . An effect size of 0.20 implies a small effect, 0.50 a medium effect, and 0.80 a large effect.

## RESULTS

**Motor behavior.** The ACL patients reproduce the target force with the same error in accuracy compared to the controls (Fig.2). ANOVA detected no main or interaction effect (between-factor group: [B1-B4]  $F_{1,16}=0.031$ ;  $p=.862$ ). The average number of reproduction repetitions was 13 in the ACL and 12 in the control group in each 3 min block.

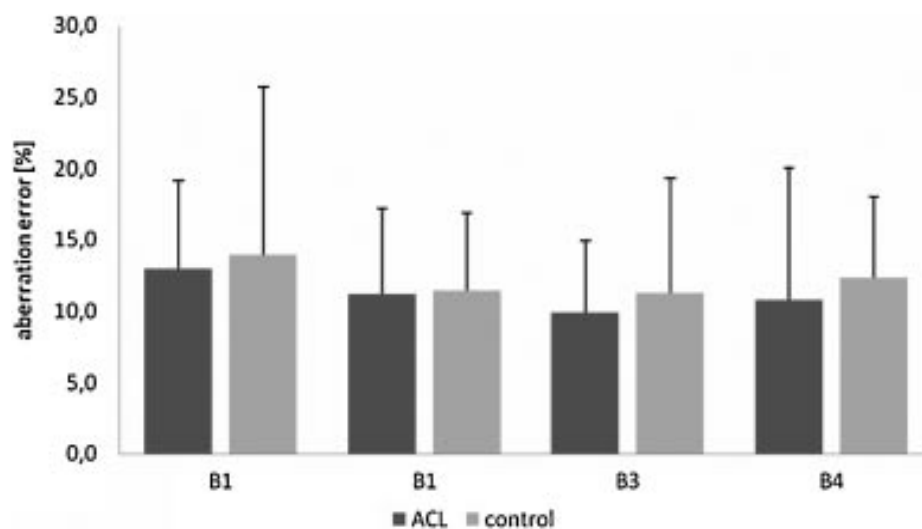


FIGURE 2. Aberration error [%] during the force reproduction task (B1-B4) in ACL patients and controls

TABLE 1. Neuromuscular activity (EMG) of each QF muscle (RF, VL, VM) measured during the force reproduction task (B1-B4) in ACL patients and controls

Muscle	N	Group	B1 (%)	B2 (%)	B3 (%)	B4 (%)
RF	9	ACL group	27.2 ± 5.0	24.8 ± 5.4	24.9 ± 4.7	24.4 ± 5.8
	9	Control group	24.9 ± 4.5	25.4 ± 6.8	24.1 ± 6.5	24.3 ± 5.8
VL	9	ACL group	32.8 ± 6.9	34.7 ± 6.1	34.6 ± 5.5	34.5 ± 7.0
	9	Control group	32.0 ± 5.5	31.9 ± 5.7	32.5 ± 6.0	32.1 ± 5.4
VM	9	ACL group	39.9 ± 6.1	40.5 ± 7.2	40.5 ± 5.9	41.1 ± 5.4
	9	Control group	43.1 ± 6.7	42.7 ± 6.8	43.4 ± 6.2	43.6 ± 6.5

TABLE 2. EEG power [ $\log \mu V^2$ ] in frontal (Theta) and parietal (Alpha-2) brain areas during the performance of a force reproduction task without visual feedback (B1-B4)

	ACL				Control				ANOVA				Post Hoc			
	B1	B2	B3	B4	B1	B2	B3	B4	(Factor/Group)				B1	B2	B3	B4
THETA frequency (frontal)																
F3	2.29 ± 0.34	2.37 ± 0.38	2.47 ± 0.36	2.41 ± 0.35	1.87 ± 0.49	1.92 ± 0.37	2.00 ± 0.58	2.03 ± 0.59	$F_{1,16} = 4.645; p = 0.047$				0.94*	1.16*	0.91*	
Fz	2.55 ± 0.37	2.60 ± 0.41	2.66 ± 0.37	2.59 ± 0.38	1.98 ± 0.46	2.00 ± 0.45	2.12 ± 0.55	2.09 ± 0.62	$F_{1,16} = 7.094; p = 0.017$				1.30**	1.33**	1.11*	0.94*
F4	2.39 ± 0.39	2.50 ± 0.44	2.56 ± 0.38	2.48 ± 0.43	1.98 ± 0.67	1.93 ± 0.49	2.07 ± 0.74	2.09 ± 0.75	$F_{1,16} = 3.355; p = 0.086$							
ALPHA-2 frequency (parietal)																
P3	1.93 ± 0.86	2.07 ± 0.80	2.14 ± 0.70	2.15 ± 0.95	2.51 ± 0.64	2.69 ± 0.72	2.71 ± 0.76	2.75 ± 0.90	$F_{1,16} = 2.702; p = .120$							
Pz	2.12 ± 0.97	2.22 ± 0.94	2.23 ± 0.81	2.26 ± 1.08	2.64 ± 0.47	2.85 ± 0.57	2.84 ± 0.61	2.82 ± 0.67	$F_{1,16} = 2.657; p = .123$							
P4	1.89 ± 0.98	2.01 ± 0.96	1.97 ± 1.01	2.05 ± 1.11	2.46 ± 0.72	2.82 ± 0.99	2.64 ± 0.84	2.81 ± 1.29	$F_{1,16} = 2.379; p = .143$							

\*\* ≤ 0.01, \* ≤ 0.05; (Post hoc values indicate hedge's d).

**EMG Parameters.** The involved EMG activity of RF, VM and VL related to the whole movement [%] showed no significant differences between controls and ACL patients (RF [B1-B4]:  $F_{1,16}=.214$ ,  $p=.650$ ; VM [B1-B4]:  $F_{1,16}=1.234$ ;  $p=.203$ ; VL [B1-B4]:  $F_{1,16}=.498$ ;  $p=.490$ ; Tab.1) and within groups.

**EEG Parameters.** A comparison of all EEG parameters in the baseline resting state before task performance between both groups demonstrates no differences. But related to the force reproduction task EEG power spectra demonstrate significant differences between ACL patients and controls in the Theta frequency band, but not in Alpha-2 spectral power (Tab.1). Figure 3 shows a descriptive demonstration of spectral Power in the Theta frequency band by showing a glow mode. This picture clearly demonstrates a higher frontal activation in the ACL patients compared to the controls indicated by brighter colours (in this case yellow).

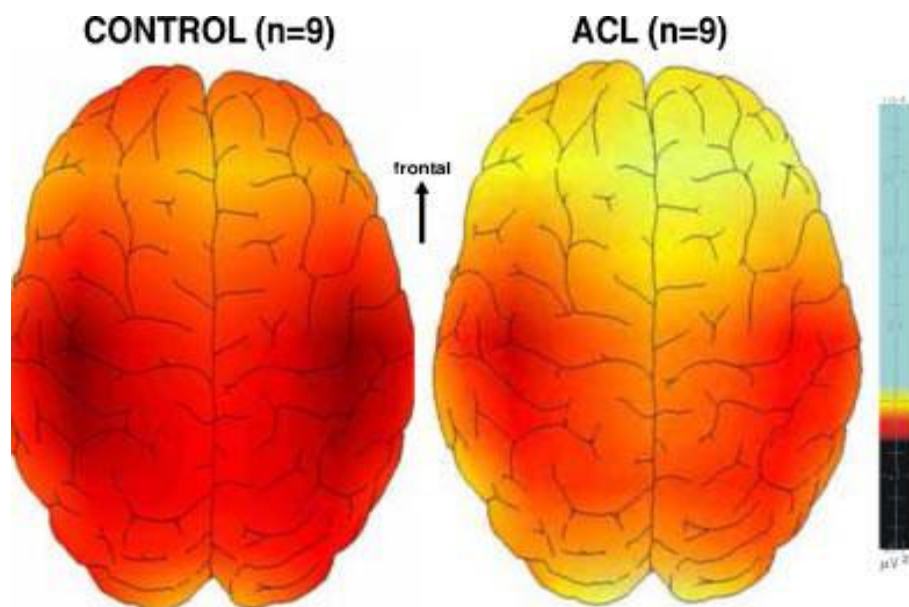


FIGURE 3. Glow modes of spectral Theta power in ACL patients and controls. Brighter colors indicate increased activity, dark colors demonstrate decreased activity (glow mode: based on spectral power of 18 real and 99 calculated electrode positions)

The statistical analysis supported the descriptive finding and demonstrated higher **Theta** spectral power in the ACL group at F3 ( $F_{1,16}=4.645$ ,  $p=.047$ , post hoc see Tab.1) and Fz ( $F_{1,16}=7.094$ ,  $p=.017$ , post hoc Tab.1). But there were no significant within-subject differences



detectable (all electrode positions:  $F_{3,48} \leq 1.146$ ,  $p \geq .107$ ). No significant results could be detected in Alpha-1 spectral power values (all electrode positions: **Alpha-1** power between-factor group  $F_{1,16} \leq 1.724$ ,  $p \geq .208$ , within-factor time  $F_{3,48} \leq 2.536$ ,  $p \geq .068$ ) **Alpha-2** power demonstrated no main effect neither between groups (all electrode positions between-factor group  $F_{1,16} \leq 2.702$ ,  $p \geq .120$ ) nor within groups ( $F_{3,48} \leq 2.561$ ;  $p \geq .120$ ). However, parietal Alpha-2 values show differences between ACL patients and controls, but not at a significant level (Tab.1). Furthermore ANOVA detected no differences in the **Beta-1** frequency band (between-factor group  $F_{1,16} \leq .648$ ,  $p \geq .433$ , within-factor time  $F_{3,48} \leq 1.892$ ,  $p \geq .144$ ).

## DISCUSSION

The present study compared precision, neuromuscular and cortical activity during a force matching task in ACL reconstructed patients and healthy controls. There were no differences in task accuracy and neuromuscular activity between both groups, but the ACL patients perform with changes in cortical activation pattern. The EEG results clearly indicate the increased frontal Theta power as the main finding in cortical activation differences.

The accuracy of force control is important for human movement. Whereas the ability of maximal force production had been investigated extensively in ACL patients the precise control of forces had not gain much interest in ACL research up to now.

The results of this study demonstrate that an ACL-reconstruction had no influence on the motor behavior associated with the accuracy in force matching performance. The participants reproduced the 50% MVIC threshold without visual feedback with an aberration error at 10-13%. This seems to be very high for a "precise" force control. This relatively high mean aberration error was supported by West et al.<sup>15</sup> who found an aberration error of 10.9% in a naive reproduction of 50% MVIC in healthy active participants comparable in anthropometric data and experimental setting to those of our study. It can be argued that the high aberration error may reflect the situation of task novelty and complexity for both groups. The only study which investigated force sensation in ACL patients was conducted by Heroux et al.<sup>11</sup> who

found a decreased performance in an easy weight discrimination task in ACL patients compared to controls. They discussed the significant reduction in precision with a loss of ligamentous sensory innervations. Therefore it seemed that a complex reproduction task used in the current study is not able to distinguish between both ACL patients and controls.

Complex neuromuscular control strategies appear to be responsible for the ability to control forces in a matching task. These strategies can be demonstrated by the activity of different muscles (in this case RF, VM, VL) related to their sum of activity. In contrast to findings of shifting EMG patterns after ACL injury or reconstruction<sup>1</sup> the results show no evidence that a different neuromuscular strategy was existent in ACL patients. The distribution of muscle activity was the same in both groups with the highest activity in the M. vastus medialis followed by M. vastus lateralis and M. rectus femoris.

Motor output and neuromuscular activity did not seem to support the concept that altered afferent proprioceptive information in ACL patients may cause changes in motor behavior. However, the results in brain activity clearly show that a reconstruction of the ACL lead to cortical changes. These changes were most prominent in the frontal Theta frequency with increased power values in the ACL group compared to the controls.

The Theta frequency is an EEG signal in the range from around 4-7 Hz described as prominent in frontal regions of the cortex. In the last decade human frontal Theta activity had been consistently described to play a major role in working memory functions including memory processes, information processing and attention in cognitive and sensorimotor tasks.<sup>16,17</sup> There is electrophysiological evidence that this signal is generated in the anterior cingulate cortex (ACC)<sup>18,19</sup>.

The ACC is thought to be part of the human attentional system involved in target selection, error detection and performance monitoring.<sup>20</sup> In a recent fMRI investigation Tana et al.<sup>21</sup> identify the ACC as the largest activation cluster in cerebral activation patterns during a sustained attention task. This is consistent with the results from other neuroimaging

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studies,<sup>22</sup> a monkey model<sup>23</sup> and brain lesion studies<sup>24</sup> suggesting the ACC as a part of the human attentional control system.

In an expanded explanation Sauseng et al.<sup>25</sup> described the dynamics of the frontal Theta frequency as a general brain integrative mechanism which is relatively specific for control of working memory processes. The general concept of working memory suggests that a limited capacity system which temporarily maintains and stores (sensory) information, supports human thought processes by providing an interface between perception, long-term memory and action<sup>26</sup> and relies on a functional network linking frontal and parietal regions of the brain. In addition to the described frontal component (Theta), the “fast” Alpha frequency (Alpha-2: 9.75-12.5 Hz) in the parietal cortex seemed to be sensitive to the amount of somatosensory information processing with spectral power values inversely related to the activation of neuronal populations in this brain area.<sup>16</sup>

The current study demonstrates significantly higher frontal Theta spectral power values in the ACL patients compared to the healthy control group. Alpha-2 power demonstrated higher values in the ACL group but do not reach significance. High individual variances in Alpha-2 may be responsible for this result.

Increased frontal Theta power associated to the ACL reconstruction may reflect a higher focussed attention and therefore a higher neurocognitive involvement during the force matching task. Although the ACL patients were able to reach the same results in force reproduction compared to healthy counterparts they have to use more neurocognitive resources related to working memory control. Altered afferent sensory information due to peripheral proprioceptive deficits after reconstruction may not match the expected information from long-term memory in the brain, which may lead to an increased load of the working memory processes.

To our knowledge only two studies have investigated altered brain activity in ACL patients. Baumeister et al.<sup>10</sup> used the same methodological approach in a joint position matching task and described changes in working memory processing including increases in frontal Theta

spectral power and decreases in parietal Alpha-2 power after ACL reconstruction. They argued that a loss or damage of mechanoreceptors in the knee joint changes the peripheral afferent sensory information which lead to an altered processing in the brain and therefore to electrocortical alterations indicating modifications in the CNS.

In a recent neuroimaging (fMRI) study Kapreli et al.<sup>12</sup> demonstrated that patients with an ACL deficiency had a changed activation especially in several sensorimotor brain areas compared to a control group.

Both studies were hardly comparable because of the different conducted tasks and methodological approaches, but they both show modifications in the brain after ACL deficiency or reconstruction and support the results of the current study. More research is needed to elucidate the detailed central mechanisms.

The findings of this study may help to establish the idea that the treatment of a peripheral injury (e.g. ACL-deficit/reconstruction) should not only focus on functional muscle deficits but even on changes in the brain as a consequence of the injury. The understanding of the underlying mechanisms of injury and cortical changes may have the possibility to optimize therapy strategies after such injuries. It would be an attractive concept to take advantage of the brains ability to change to find strategies to strengthen the results of rehabilitation with applications in patient's re-education and training.

Nevertheless two limitations should be considered when drawing interpretations from these findings. First, the study demonstrated increased frontal Theta power in ACL patients during a sensorimotor task and statistics support the claim by significance and effect sizes that the observed differences are real. There is only indirect evidence of a correlation to attention or working memory control. Further studies should try to evaluate attentional control parameters to strengthen the relationship. Secondly, no functional testing to assess further similarities or dissimilarities between ACL patients and the control group was included which would have strengthened the results. An expanded protocol would have support the interpretation of

cortical changes related to neurocognitive compensation or impairment. This should be applied to further research.

The results of this study demonstrated clearly that neurocognitive functions have changed after ACL reconstruction indicated by frontal Theta spectral power measured by EEG whereas performance and neuromuscular activity were not altered. The results suggest that an ACL reconstruction may lead to cortical changes in relation to the control of working memory processes.

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## PUBLICATION 10

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## ABSTRACT

Exercise-induced-muscle-damage (EIMD) is characterized by pain, swelling and shortening of the muscle, increased serum creatine kinase, decreased force output and altered neuromuscular function. The aim of this study was to investigate the effects of EIMD to determine the relationship between the peripheral symptoms, neuromuscular changes and delayed pain sensation during a submaximal movement of the biceps brachii on cortical alpha ( $\alpha$ ) activity. In contrast to the control (n=12) group, the experimental (n=16) group participated in an EIMD protocol and both groups were monitored for 132h post-EIMD protocol. Muscle pain scores in the experimental group peaked after 36h with the lowest torque reported at 12h.  $\alpha$ -1 activity increased significantly in the motor and somatosensory area 12h post-EIMD while  $\alpha$ -2 activity increased in the contralateral fronto-central area. At 12h neuromuscular functioning was already disturbed while the sensation of pain was perceived but not fully developed. At 36h pain had further increased and neuromuscular function improved whilst  $\alpha$ -1 and  $\alpha$ -2 activities had decreased. We hypothesise that  $\alpha$ -1 activity over the motor and somatosensory cortex of the experimental group displays a compensatory increase in response to the changes in neuromuscular function during movement, whilst an increase in  $\alpha$ -2 activity is related to the suppression of pain experienced within the first 12h.

**Keywords:** EIMD, muscle pain, cortical alpha activity, central regulator, top-down control, EEG



## INTRODUCTION

Exercise induced muscle damage (EIMD) is a phenomenon which occurs after unaccustomed exercise, especially exercise where the muscles are lengthened under load. Symptoms include structural damage to the muscle (Fridén and Lieber, 1992), development of pain (Plattner et al., 2011) and changes in neuromuscular function including electromyography activity (EMG) (Plattner et al., 2011; Semmler et al., 2007) and force production (Semmler et al., 2007).

Interestingly whilst there is a decrease in EMG activity during a maximal voluntary contraction, submaximal EMG is increased within the first 12 h after an EIMD protocol. This is of interest since the sensation of pain is only perceived 12- 24 h after an EIMD protocol (Plattner et al., 2011). Collectively these data suggest that the changes observed in the neuromuscular function after EIMD are driven by an upstream regulator situated in the brain, which is guided by peripheral bottom-up input (i.e symptoms of pain, inflammation, intramuscular enzyme leakage and tissue damage) (Plattner et al., 2011; Semmler et al., 2007).

It has been shown with fMRI and PET scans that a pain stimulus activates several areas of the brain, the primary somatosensory cortex (SI), secondary somatosensory cortex (SII), premotor cortex and cingulate gyrus (limbic system) contralateral to the stimulus (Vogt, 2005). These areas integrate the information arising from the periphery to create an emotional, behavioural and/or motor response, depending on the circumstances (Melzack, 1999; Vogt, 2005).

As fMRI does not allow for movement during the measurement or precise time resolution of the data (Sato et al., 2010), electroencephalography (EEG) is a preferred method to measure brain activity during movement (Thompson et al., 2008). EEG is measured on the scalp with multiple low-amplitude sensitive electrodes and measures electrical activity produced by the neuronal firing in cortical and subcortical areas of the brain (Niedermeyer E, 2005; Thompson et al., 2008). An outcome of the EEG measurement is spectral power in different

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frequencies also known as frequency band activity. The 8-13Hz band is known as the alpha ( $\alpha$ ) activity, which is known to be influenced by pain (Chang et al., 2001; Chang et al., 2003), movement (Stancak, Jr. et al., 2000), attention (Sauseng et al., 2006) and arousal (Chang et al., 2005).

The most common response of  $\alpha$  activity to a painful, cognitive or motor stimulus is a decrease in activity at the onset of the stimulus, followed by an increase in activity once the stimulus has lasted for several minutes (Chang et al., 2003; Stancak, Jr. et al., 2000) or ended (Anderson and Ding, 2011; Chang et al., 2001). However recent research has shown that  $\alpha$  activity can also increase during focused attention and cognitive processes (Palva and Palva, 2007a). Von Stein (2000)(von et al., 2000) showed that the increase in  $\alpha$ -activity especially  $\alpha$ -2 activity was due to interactions between the frontal and parietal areas and that the increase appears to be acting as a top down regulator. A similar top down regulatory process has been suggested by Chen and Herrmann (Chen and Herrmann, 2001a) to control the sensation of pain. The notion of the top down suppression of pain is further supported by studies showing that meditation increases the global  $\alpha$ -activity whilst decreasing the subjective sensation of pain (Kakigi et al., 2005; Lagopoulos et al., 2009).

EIMD provides an interesting model for investigating the effect of structural damage and acute pain on  $\alpha$ -activity, particularly in the first 12 h after EIMD when neuromuscular changes occur with minor symptoms of pain (Plattner et al., 2011). Therefore the aim of this study was to investigate the effects of EIMD and determine whether there is a relationship between the peripheral symptoms, the neuromuscular changes and delayed pain sensation and cortical  $\alpha$ -1 and  $\alpha$ -2 activity.

We hypothesise that a compensatory increase in  $\alpha$ -1 activity will be displayed over the motor and somatosensory cortex of the experimental group compared to the control group in response to changes in neuromuscular function. We further hypothesize that an increase in  $\alpha$ -2 activity is associated with the suppression of the sensation of pain experienced within the first 12 h after the EIMD inducing protocol was performed.

## METHODS

Thirty-seven right-handed male participants, aged 21-40 years, were recruited for this study. Handedness was determined by the *Edinburgh* handedness inventory (Oldfield, 1971). Participants matched for age, height, weight, body fat and skinfold thickness, were allocated to the experimental or control group. All participants had to be free of any upper body injuries and were not participating in any upper body training during the last 12 weeks before the study. This included the engagement in exercises involving specific muscle lengthening under tension movements.

TABLE 1. Descriptive data for the controls (n=12) and experimental group (n=16)

Variable	Control	Experimental
Age (years)	23 ± 4	23 ± 3
Body mass (kg)	71.1 ± 8.8	72.7 ± 11.3
Stature (cm)	171.7 ± 6.8	177.4 ± 8.0
Body fat (%)	15.9 ± 4.9	13.4 ± 5.4
Skinfolds (mm)	79 ± 37	69 ± 38
Handedness (%)	73 ± 20	79 ± 19

Data are expressed as mean ± standard deviation.

Prior to being informed about the study design and familiarization with the equipment participants had to sign an informed consent form and complete a Physical Activity Readiness Questionnaire (Par-Q) (American College of Sports Medicine, 2007). They were also asked to fill out questionnaires about their injury and training history. Participants were informed about the study design, familiarized with the equipment and signed the consent form before starting the study. The study was approved by the Human Ethics Committee of the Faculty of Health Science, University of Cape Town. The principles outlined by the Declaration of Helsinki for the use of Humans were adopted in this study (, 2002).

### Study design

Before the start of the study all participants were familiarized with the testing equipment and different test protocols. Figure 1 is a time line depicting the order of tests performed over the

seven day testing period. To minimize the effect of circadian rhythm on any of the outcome measures, all tests were scheduled at the same time of the day (within 60 min). This however was not possible for the measurement at 12 h after the exercise protocol.

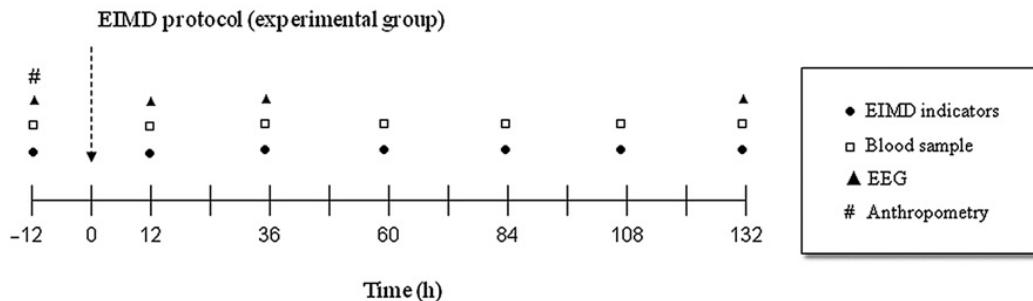


FIGURE 1. Timeline of measurements. The EIMD indicators include, pain, arm circumference, elbow angle, creatine kinase activity, EEG

Twelve hours before the start of exercise protocol (see also Figure 1), stature, body mass, body fat percentage and skinfolds of each participant was measured. In addition resting elbow angle, elbow muscle function (maximal voluntary contraction), biceps girth and pain scores were measured. A blood sample was taken to determine creatine kinase activity.

Electroencephalographically activity (EEG) was measured during a self-initiated self paced flexion-extension movement. In contrast to all the above mentioned measurements which were conducted at -12, 12, 36, 60, 84, 108 and 132 h, EEG measurement were only captured at -12, 12, 36 and 132 h (Figure 1). These measurements were time consuming for the participants and there was concern about poor compliance if they were required to be tested more frequently.

### Exercise protocol

Twelve hours after baseline testing, the subjects in the experimental group completed an exercise protocol designed to induce muscle damage (EIMD protocol). In brief, participants were asked to resist the lengthening movement of the left biceps (5 sets of 25 movements; see also 'muscle function tests' section for set up of the Biodex (Biodex pro 3, New York,

USA)). The resistance to these movements was set on a Biodex dynamometer at 80% of each subject's maximum isometric contraction torque, as this has been shown to induce EIMD (Plattner et al., 2011). The control group did not perform this exercise protocol.

### **Muscle function tests**

The muscle function tests consisted of a maximal voluntary contraction (MVC) measurement and a self-paced submaximal flexion-extension movement. The MVC was measured using a Biodex dynamometer when performing elbow flexion of the left arm. For this, the participant sat in the chair of the Biodex dynamometer with their upper body and left upper arm securely strapped to the dynamometer, while the left forearm was only able to move in the sagittal plane (flexion/extension). In this position, participants were able to freely flex and extend their elbow over a range of approximately 120°, without hyper-extending the elbow. The rotation axis of the dynamometer was aligned with the lateral epicondyle of the humerus, while the forearm was fixed into a fully supinated position. This ensured that the flexion/extension movement was carried out in the transversal axis and sagittal plane. Participants were asked to perform three 5 s isometric elbow contractions at maximal effort, with a fixed dynamometer arm angle of 45°. This set-up will result in an elbow angle at about 60°, which is within the optimal length tension curve range (Chang et al., 1999; Saxton and Donnelly, 1996). Participants were asked to perform three 5 s MVC's interspaced by 60s recovery periods, as previously described (Plattner et al., 2011).

The flexion-extension movement was not performed on the Biodex, but rather the participants were seated on a standard armless-chair. Their arms were relaxed and hanging by their sides. For the submaximal self paced flexion and extension movements all participants wore a 1kg wrist strap and movements were performed in the sagittal plane between elbow angles of 180° and 90°. During the movements subjects were ask to look at a fixed point at the wall to reduce the interference of eye movements on the EEG measurement (see also EEG evaluation). In addition, the upper body and upper arm were

positioned as described in the MVC set-up for standardization purposes. Participants were asked to perform 75 repetitions, which were interspaced by 5-10s recovery periods with slightly longer rest periods after each 25 repetitions, while EEG data were captured.

The experimental as well as the control group had to perform these muscle function tests.

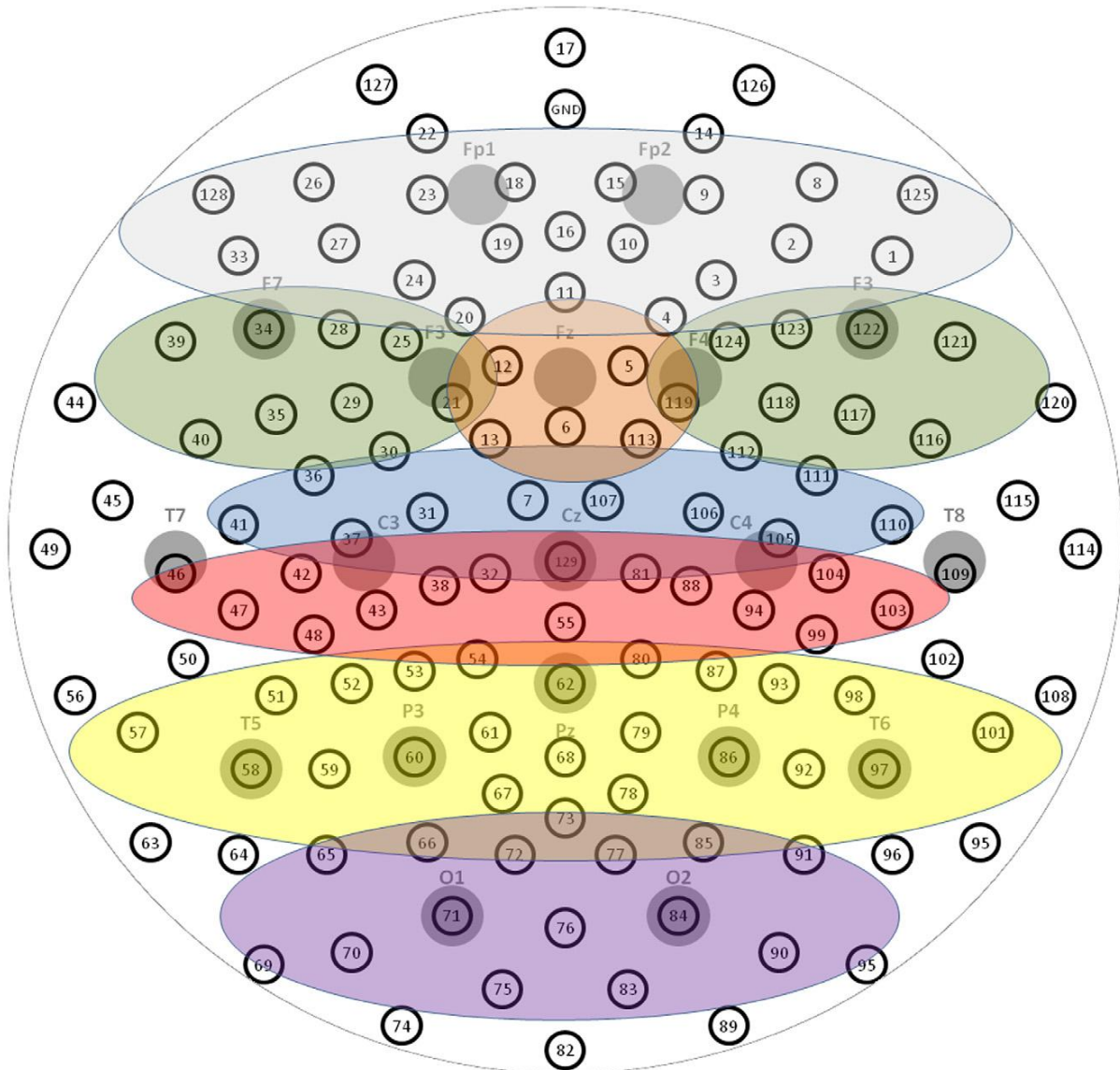


FIGURE 2. A layout of the EGI 129 channel system overlaid by the 10:20 electrode system (dark grey circles). Ellipses represent the following gross cortical areas: Grey (Frontal), Green (Premotor), Orange (Supplementary Motor), Blue (Motor), Red (Somatosensory), Yellow (Parietal), Purple (Occipital).

### **Electroencephalographic study procedure**

The EEG data were obtained in a darkened, sound-attenuated, temperature controlled room to minimize the effect of confounding factors. Participants were instructed how to perform the self paced flexion and extension movements. EEG activity was measured during the 75 submaximal fast self paced flexion and extension movements. In addition, subjects were asked to keep their eyes open and focused on a fixed spot on the wall during the submaximal self paced movements.

### **Electroencephalographic recording**

An EEG net with 128 recording sites plus a vertex reference electrode (electrode 129) Electrical Geodesic™ system (Electrical Geodesics, Inc, Oregon, USA) (Bernier et al., 2007), which measures the electrical activity on the surface of the scalp, was fitted onto each participant (see Figure 2 for an electrode layout). The impedance of all electrodes was maintained below 50 k $\Omega$  as suggested by the manufacture of the EGI system and different technical references (Bernier et al., 2007; Keil and Muller, 2010; Murias et al., 2007) due to the high input resistance of the EEG amplifier. Specially designed amplifiers processed the high impedance signal. EEG was recorded using a 0.1–50 Hz bandpass filter (3 dB attenuation) (Murias et al., 2007). The signals were sampled at 250 Hz (Bernier et al., 2007; Murias et al., 2007). All recordings were initially referenced to the central reference electrode (Cz/129) (Bernier et al., 2007; Murias et al., 2007). The EEG system was connected to an experimental workstation (Net Station software, Apple Inc desktop) (Bernier et al., 2007).

### **Electroencephalographic data analysis**

The raw EEG data was 50 Hz notch filtered as well as 1-40 Hz band pass filtered. Following this the data for each trial were segmented into 3s epochs [58]. Data were collected continuously during the premovement, movement and rest phase and therefore represent the

common state of the brain during a biceps brachii movement protocol rather than a movement induced change.

The EEG was re-referenced against an average reference (Bernier et al., 2007; Bertrand et al., 1985; Keil and Muller, 2010), which made data recorded in the reference electrode (Cz/129) available for analysis. The EEG recording was monitored for movement, eye movement, blink artefacts, and noise by an amplitude threshold criterion. Epochs were excluded if the eye blink threshold exceeded 140  $\mu\text{V}$  and the eye movement threshold exceeded 55  $\mu\text{V}$ . Ocular artifacts were also detected by a slope threshold, if the slope of a channel increased more than than 14  $\mu\text{V}/\text{ms}$  that channel was discarded within that epoch. The amplitude of a given channel was excluded in that epoch if it exceeded  $\pm 150 \mu\text{V}$ , and in such a case replaced by an estimate calculated of the values of the surrounding channels. This was performed by computer algorithms built into the Net Station software version 4.2.3, (Electrical Geodesics, Inc, Oregon, USA) (Murias et al., 2007).

After the automated artefact rejection algorithm all epochs were also visually inspected offline and the recordings in which artefacts could not be removed were manually discarded before further data analysis took place.

While shorter epochs might reveal the relationship between, for example, spectral estimates or time-locked events, our intent was to characterize background brain states (induced activity) rather than specific components of event related processing (Travis et al., 2002). Subjects who had less than 40 usable, artefact-free epochs per trial day were excluded from further analysis. A Fast Fourier Transform (FFT) with a Welch window was performed to obtain the spectral information of an epoch of each subject. Hereafter the data was exported to Microsoft Excel<sup>®</sup> and each subject's data were averaged before further analysis.

The different frequency bands used in this study were as follows:  $\alpha$ -1 (7.81-9.77 Hz) and  $\alpha$ -2 (10.74-12.7 Hz). Thereafter the relative power (activity) for each frequency on each day was calculated with the following formula:  $((\text{Power (12h or 36h or 132h)} - \text{Power (0h)}) / \text{Power (0h)}) * 100$ . The different relative power values for each subject on the different testing days



were used to calculate the statistical differences between the two different groups on the four different testing days.

Matlab 6.5 (The Mathworks Inc., Massachusetts, USA) and EEGLab v 5.02 (SCCN, University of California, San Diego, USA) were used to create topographical maps of the relative power on each day in each frequency.

Recorded data are represented based on the 10:20 system. All electrodes are grouped according to electrode on the 10:20 system which represents the same area. For example the 10;20 electrode Fz is represented by electrodes 5,6, 11 and 12 in the Netstation system (Figure 2).

### **Other measurements**

Blood samples, biceps girth, resting elbow angle and a pain score were measured daily as previously described by Plattner et al. (2011). For the blood sample 5 ml of blood were drawn from the right antecubital vein. These samples were stored (-20° C) and later analyzed to determine the serum creatine kinase (CK) activity in the blood (Beckman DU-62, Beckman Instruments, Fullerton, California, USA) as described previously (Lambert et al., 2002). The girth of the left biceps was measured with a tape measure midway between the acromion and radial bony landmarks, that was marked with a permanent marker for repeatability purposes (Lambert et al., 2002). Resting elbow angles, and by implication the resting length of the biceps muscle were measured with a goniometer (Lambert et al., 2002). Current pain perception was measured on a daily basis before the muscle function test with the use of a 10 cm visual analogue scale (VAS) (Svensson et al., 1998).

### **Statistical analysis**

An independent t-test was used to compare the descriptive data between experimental and control group, using STATISTICA 8.0 data analysis software (StatSoft, Inc. Tulsa, OK, USA).

As some of the data sets in this study had an unequal variance, determined using Levene's test of homogeneity of variance, it was decided to use non parametric statistical tests instead of the parametric ANOVA test. A Kruskal-Wallis test (H) compared the differences between the control and experimental group on each of the testing days in each electrode separately. A Friedman's test ( $X^2$ ) was used to compare changes within each group over the repeated testing days in each electrode separately. A Dunn's test was used for *post-hoc* analysis. Statistical significance was accepted at  $p < 0.05$ .

## RESULTS

### Characteristics of subjects

One participant did not finish the entire trial and was excluded from the study. Seven other participants were also excluded because they did not have sufficient EEG data epochs for further analysis. The remaining twenty eight participants were divided into two groups similar in weight, height, age, skinfold thickness and handedness (Table 1).

### Muscle soreness

The difference in pain in the left arm in the experimental and control group measured by the means of the VAS scale is shown in Figure 3a. Peak pain in the experimental group was observed 36 h after the EIMD inducing protocol ( $X^2 = 53.66$   $p = 0.00001$ ). A difference in pain between the two groups can be observed at 12 (H = 7.48  $p = 0.0062$ ), 36h (H = 14.32  $p = 0.0002$ ), 60 (H = 10.21  $p = 0.0014$ ), 84 (H = 8.03  $p = 0.0046$ ) and 108 h (H = 8.37  $p = 0.0038$ ). Significant changes in pain occurred in the experimental group compared to the baseline value at 12, 36, 60 84 and 108 h ( $X^2 = 53.66$   $p = 0.00001$ ) (Figure 3 a).

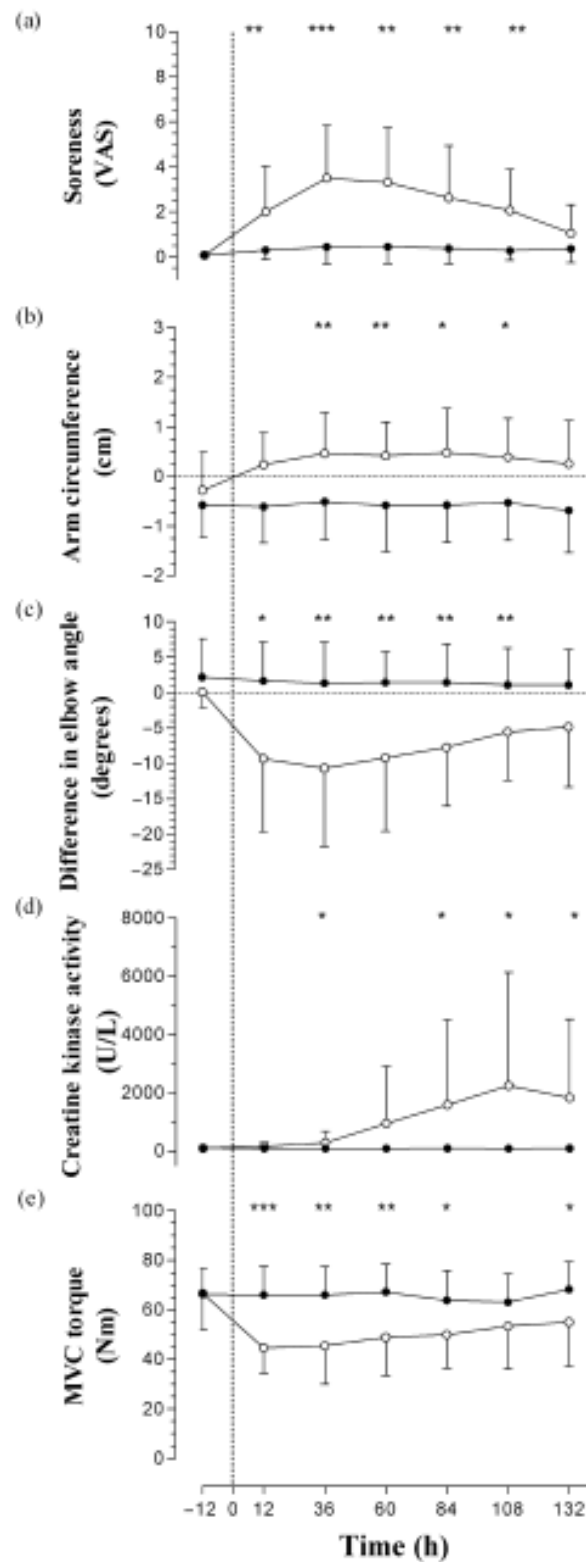


FIGURE 3. (a) The change in current pain measured with the visual analogue scale (VAS). (b) The change in the difference in relaxed elbow girth (cm) between the left and right arm. (c) The change in the difference in elbow angle (degrees) between the left and right arm. (d) The change in creatine kinase activity (U/L). € The maximal force output produced on seven consecutive days. \* Indicates results of Kruskal-Wallis nonparametric test in the control (●) and experimental (○) group on seven consecutive days. \* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$

### **Arm circumference**

During the experiment there was a significant increase in the difference in girth between the exercised and rested arm in the experimental group compared to the control group at 36 (H = 7.23 p = 0.0072), 60 (H = 6.97 p = 0.0093), 84 (H = 5.36 p = 0.0207) and 108 h (H = 5.04 p = 0.0248). Significant changes in arm circumference were also found over time in the experimental group compared to the baseline value at 36, 60 and 84 h ( $X^2 = 27.04$  p = 0.0001). The difference between the left and right biceps girth of the control group did not change throughout the experiment. The difference between the left and right biceps girth of the control group did not change throughout the experiment (Figure 3 b).

### **Resting elbow joint angle (muscle length)**

The difference in resting joint angle between the left and right arms in the control and the experimental group are shown in Figure 3c. There was a significant decrease in elbow joint angle in the experimental group until 84 h after the EIMD protocol ( $X^2 = 42.46$  p = 0.0001). The difference in joint angle decreased in the experimental group compared to the control group and reached its minimum 36 h (H = 7.34 p = 0.0067) after the exercise protocol. It remained decreased until 108 h (H=6.71 p=0.0096) after the exercise protocol. No changes in the resting elbow joint angle over time were observed in the control group (Figure 3 c).

### **Serum CK activity**

The serum creatine kinase activity in the experimental group increased at 36 h (H=3.90 p=0.0484) in the experimental group and reached its highest values compared to the control group at 84 (H=3.99 p=0.0456), 108 (H=4.87 p=0.0274) and 132 h (H=5.27 p=0.0217) after the EIMD protocol. Creatine kinase levels in the experimental group were only significantly increased compared to baseline at 108 and 132 h ( $X^2 = 20.27$ , p = 0.0025). The serum

creatine kinase activity in the control group did not change during the experiment (Figure 3 d).

### **Muscle function**

Muscle function, as measured by MVC (Figure 3e), decreased significantly in the experimental group compared to the control group on all but one visit to the laboratory after the EIMD inducing protocol ( $p < 0.05$ ). The largest decrease in maximal force output was observed within the first 12 h after the EIMD protocol in the experimental group ( $H = 14.14$   $p = 0.0002$ ) while there were no changes in the control group throughout the experiment. The force output in the experimental group remained different to that of the control group until the end of the trial ( $H = 5.61$   $p = 0.0179$ ) (Figure 3 e).

A difference was also observed in the force output of the experimental group over time at 12, 36, 60 and 84 h compared to the baseline measurement ( $X^2 = 48.3$   $p = 0.0001$ ). There were no changes in the control group over time.

### **Alpha 1**

Figures 4 and 6 show significant differences between the control and experimental group at 12 and 36 h after the exercise protocol. The most increases are seen 12 h after the exercise protocol, although changes remain at 36 h especially in the electrodes overlying the motor and somatosensory areas (Cz, C3, and C4).

At 12 h after the EIMD protocol, there was a widespread increase in  $\alpha$ -1 power over the motor and somatosensory area in the experimental group compared with the control group. This is seen especially at electrodes representing the central motor area surrounding C3, C4, and Cz.

Thirty-six hours after the exercise protocol, differences were still observed between the experimental and control groups, especially in the electrodes overlying the medial motor area.

To simplify the understanding of the results, electrodes have been placed into subgroups and labeled with the title of the closest electrode represented on the 10:20 system.

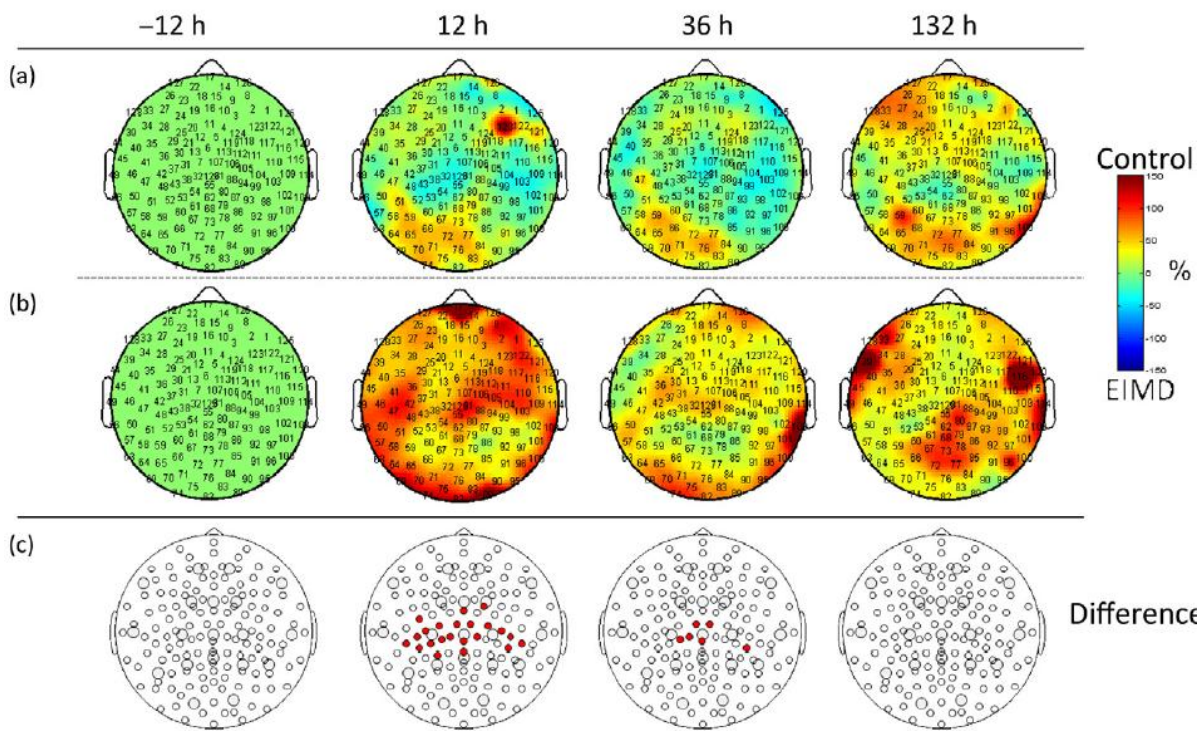


FIGURE 4. The global change (%) of  $\alpha$ -1 activity measured with 129 electrodes over the scalp is shown in the control (a) and experimental (b) group. An outline of the electrodes showing significant differences between the two groups (c) at each time point is also shown.

## FRONTAL

12h

*Fz and F4.* Electrodes 6 and 113 ( $H > 4.56$   $P < 0.05$ ) placed on the frontal area of the cortex show significant differences between the groups.

36 and 132h

No changes were seen in electrodes over the frontal area 36 or 132 h after the protocol, which induced the muscle damage.

## CENTRAL

12h

T3. A significant differences was found in electrode 47 ( $H = 4.56$   $P < 0.05$ ), which is located between C3 and T3.

C3. In the ipsilateral central area surrounding electrode C3 significant differences were seen in electrodes, 31, 32, 36, 37, 42, and 48 ( $H > 4.17$   $P < 0.05$ ), and electrodes 38 and 43 ( $H > 7.76$   $P < 0.01$ ) where  $\alpha$ -1 activity increases were significant in the experimental vs the control group.

Cz. Significant changes between the two groups were also seen over the vertex (Cz) of the head in electrodes 7 ( $H = 6.28$   $P < 0.05$ ) as well as 81 and 129 ( $H > 7.76$   $P < 0.01$ ).

C4. Significant differences were also seen in the central area contralateral to the moving arm in electrodes 99 and 105 ( $H = 5.61$   $P < 0.05$ ) as well as 104, 106, and 111 ( $H > 7.00$   $P < 0.01$ ).

36h

Cz. The largest differences were recorded in the three electrodes surrounding the vertex area, electrodes 7, 107, and 129 ( $H > 4.17$   $P < 0.05$ ).

C3. Differences occurred between the experimental and the control group at electrodes 32 and 38 ( $H > 4.76$   $P < 0.05$ ) in the motor area ipsilateral to the movement (between C3 and Cz).

C4. Differences occurred between the groups in electrode 99 ( $H = 6.76$   $P < 0.01$ ).

132h

No differences were observed in a-1 activity between the two groups at 132 h.

## PARIETAL AND TEMPORAL AREAS

12h

*P3 and Pz.* There were significant differences in electrode 53 ( $H = 3.99$   $P < 0.05$ ), representing P3, and also in electrode 55 ( $H = 8.28$   $P < 0.01$ ) representing Pz.

*T4.* Further down in the temporal areas, there was a significant difference between groups (electrode 103;  $H = 7.77$   $P < 0.01$ ).

36 and 132h

No differences were observed in a-1 activity in the parietal or temporal region between the two groups at 36 or 132 h.

## Alpha 2

Figures 5 and 7 show that there are significant differences in the a-2 activity between the control and experimental groups at 12 and 36 h after the exercise protocol.

## FRONTAL

12h

*F3.* In the ipsilateral frontal area only one electrode was significantly different, electrode 29 ( $H = 4.56$   $P < 0.05$ ).

*Fz.* In the medial frontal area, electrodes 4, 6, and 12 ( $H > 3.99$   $P < 0.05$ ) were significantly different between the two groups.



F4. In the contralateral frontal area electrodes 112, 119, and 124 ( $H > 4.76 P < 0.05$ ) as well as 113 and 118 ( $H > 7.25 P < 0.01$ ), there were significant differences between the groups while in the ipsilateral frontal area, only one electrode (29) showed significant differences ( $H = 4.56 P < 0.05$ ).

### 36 and 132h

No changes were seen in electrodes over the frontal area 36 or 132 h after the protocol, which induced muscle damage.

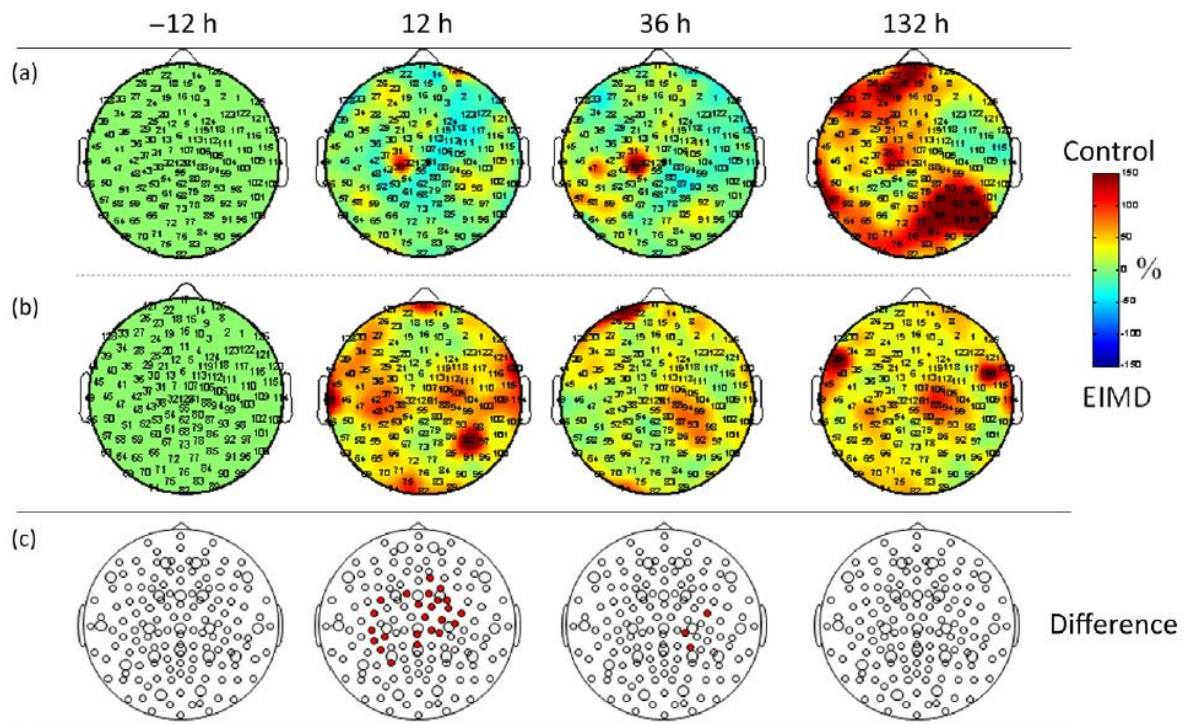


FIGURE 5. The global change (%) of  $\alpha$ -2 activity measured with 129 electrodes over the scalp is shown in the control (a) and experimental (b) group. An outline of the electrodes showing significant differences between the two groups (c) at each time point is also shown.

### CENTRAL

#### 12h

C3. Differences between groups occurred in the ipsilateral central area at electrode 42, 43 and 48 ( $H > 4.97 P < 0.05$ ).

Cz. In the medial central area at electrodes 107 and 129 ( $H > 4.76$   $P < 0.05$ ) there were differences between the groups.

C4. In the contralateral central area surrounding C4, there were differences between groups at electrodes 88, 105 ( $H > 5.17$   $P < 0.05$ ), and 106 ( $H = 11.80$   $P = 0.001$ ).

36h

C4. Significant differences between groups occurred in electrodes 88, 105, and 111 ( $H > 4.17$   $P < 0.05$ ).

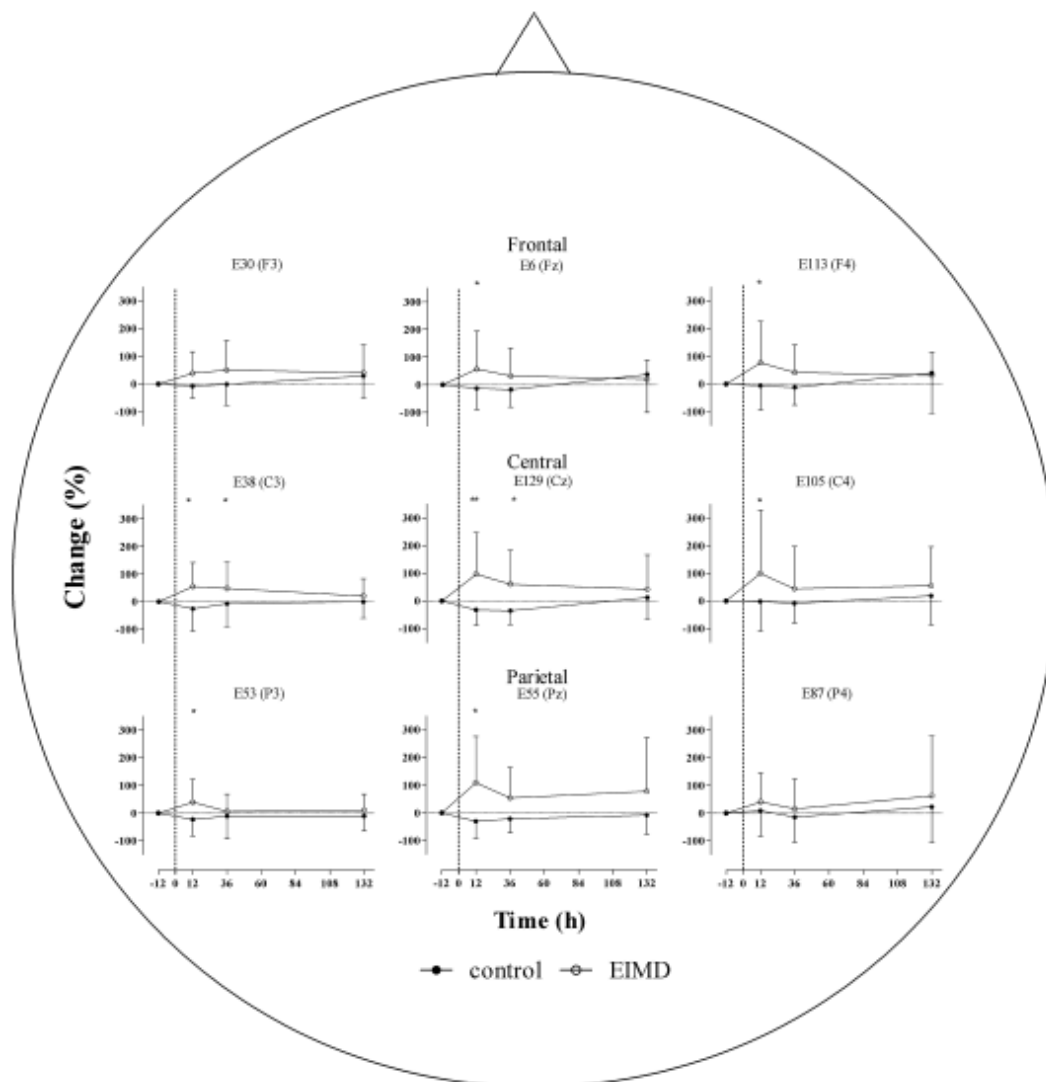


FIGURE 6. Nine different electrodes representative of the change (%) of  $\alpha$ -1 activity in the frontal, central and parietal areas of the brain. Each of the electrodes represents a location on the 10:20 system. \* Indicates results of Kruskal-Wallis nonparametric test in the control (●) and experimental (○) group on seven consecutive days. \* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$

132h

No differences were observed in the central area between the two groups at 132 h.

## PARIETAL

12h

*P3.* The difference between groups occurred in the parietal area at 52 ( $H = 7.50$   $P < 0.01$ ), and 60 ( $H = 4.17$   $P < 0.05$ ).

*Pz.* Of the electrodes representing Pz, only electrode 55 ( $H = 5.61$   $P < 0.05$ ) showed significant differences between the groups.

36h

*P4.* There were significant differences between groups in electrode 87 ( $H = 6.28$   $P < 0.05$ ).

132h

No differences were observed in the parietal area between the two groups at 132 h.

## DISCUSSION

The first finding of this study was that the EIMD protocol resulted in similar physiological responses (Figure 3) reflecting muscle damage in the experimental group as previously reported (Plattner et al., 2011).

In particular the symptoms of EIMD (swelling, muscle shortening and CK activity) changed in the typical way for the duration of the experiment (Figure 3a-d). Also, muscle function (force output) was impaired immediately after the EIMD protocol (Plattner et al., 2011; Semmler et al., 2007) and gradually recovered, but did not return to baseline by 132 h (Figure 3e). Pain on the other hand progressively increased, peaking around 36 to 60 h and was decreasing at 132 h.

The following sections will discuss  $\alpha$ -1 and  $\alpha$ -2 activity separately, as the two bands have shown to be active in separate cortical areas between the groups. The authors acknowledge that deducing functional and anatomical associations from EEG data is challenging, but they do believe that the clustering of significant differences in certain areas do support the association with areas and functionality.

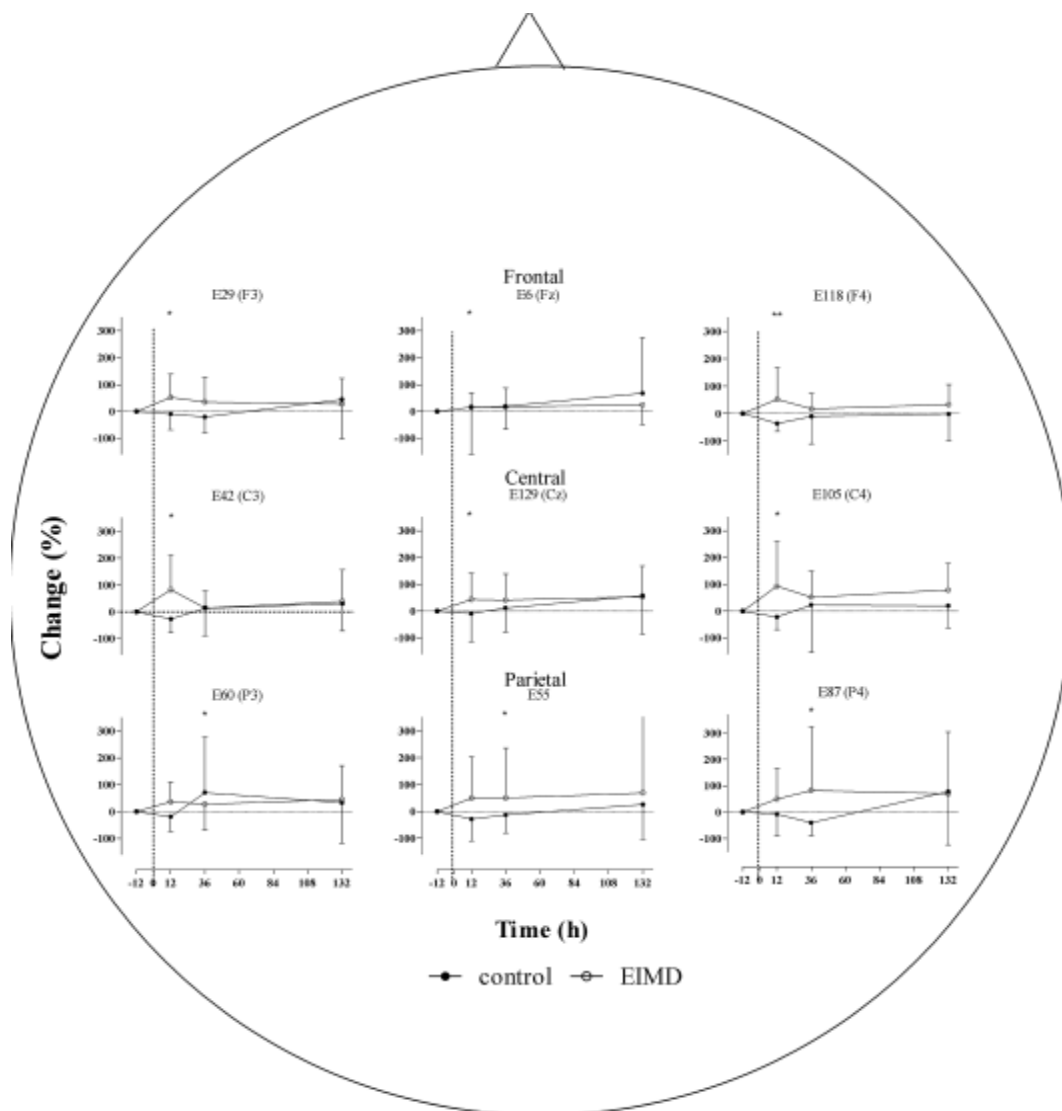


FIGURE 7. Nine different electrodes representative of the change (%) of  $\alpha$ -1 activity in the frontal, central and parietal areas of the brain. Each of the electrodes represents a location on the 10:20 system. \* Indicates results of Kruskal-Wallis nonparametric test in the control (●) and experimental (○) group on seven consecutive days. \* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$

## Alpha 1

The participants with symptoms of EIMD experienced an increase in cortical  $\alpha$ -1 activity (Figure 4 and 6) while performing the series of brisk biceps brachii contractions. The increase in the experimental group was most noticeable at 12 h post-EIMD induction and activity had decreased, although still significantly different between the two groups, by 36 h post. The activity was most pronounced in electrodes overlying the motor and somatosensory cortex (Figure 4c).

Increased  $\alpha$ -1 activity in the somatosensory and motor cortex has previously been associated with movement planning (Anderson and Ding, 2011), as well as increased consciousness and perception (Palva and Palva, 2007b). Altered perception, reflected in the increased  $\alpha$ -1 activity, might be a response to changed peripheral bottom-up feedback. Feedback to the central nervous system reflects changes in the peripheral neuromuscular function (greater movement unsteadiness, increased submaximal EMG activity and decreased force output) (Plattner et al., 2011; Semmler et al., 2007), which need to be integrated and processed.

The increase in  $\alpha$ -1 activity could also be a result of precise movement planning and execution in the motor and somatosensory cortex to compensate (feed forward) for the loss in neuromuscular function whilst experiencing the symptoms of EIMD, indicated by the increased submaximal peripheral EMG activity.

We have previously shown (using the same protocol) that submaximal EMG activity increases within the first 12 to 36 h after an EIMD protocol (Plattner et al., 2011), while maximal EMG and force output decrease. It has been suggested that the increased submaximal EMG activity is due to increased neural drive initiated by the central nervous system (McAuley et al., 1997; Semmler et al., 2007). Thus our current findings of increased cortical  $\alpha$ -1 activity in electrodes overlying the motor and somatosensory area together with the previous findings of increased submaximal and decreased maximal EMG activity at 12

and 36 h post-EIMD, support our hypothesis that the motor and somatosensory cortex act as an compensatory upstream regulator of motor control while experiencing EIMD.

While it cannot be clearly stated what causes the increases in  $\alpha$ -1 activity over the motor and somatosensory area in the EIMD group it is suggested that increased cortical  $\alpha$ -1 activity might be necessary to counteract the loss of movement steadiness and force output whilst experiencing EIMD. Due to the recording of  $\alpha$ -1 activity in this proximal location the authors assume it to be a top- down regulator of peripheral function. Therefore the increased  $\alpha$ -1 activity could be part of an upstream regulatory mechanism of motor perception, activation and neuromuscular function.

## **Alpha 2**

While  $\alpha$ -1 activity increased in the motor and somatosensory areas,  $\alpha$ -2 activity increased in the ipsilateral fronto-parietal area as well as in the contralateral fronto-central areas.

$\alpha$ -2 activity peaked at 12 h (Figure 5 and 7), when neuromuscular function was already disturbed although the main sensation of pain was yet to develop. At 36 h post-EIMD protocol  $\alpha$ -2 activity remained elevated in the contralateral centro-parietal area. Pain peaked at 36 h whilst  $\alpha$ -2 activity decreased again towards pre-EIMD values.

It has previously been shown that  $\alpha$ -2 activity increases due to interactions between the frontal and parietal cortical areas. Palva and Palva (Palva and Palva, 2007a) and Halgren et al (Halgren et al., 2002) proposed that this fronto-parietal  $\alpha$  synchrony is associated with focused attention, working memory, conscious perception, cognition and action. As this is assumed to be the most proximal level of control, this fronto-parietal  $\alpha$  synchrony is known to act as a top down regulator in subcortical and peripheral information integration processes (von et al., 2000).

Klimesch et al (2007) have further suggested that increased  $\alpha$ -2 activity in cortical areas causes an inhibition of information retrieval from the involved areas. Hence the authors suggested the increase in  $\alpha$  activity to be an inhibitory top down control mechanism of

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information integration processes. A similar cortical top down regulator has been suggested for pain (Chen and Herrmann, 2001b), stating that the painful signal is perceived and incorporated at different frequency levels and areas of the cortex, with the somatosensory and the frontal cortex playing an important part.

Further research by Kakigi and Lagopoulos (Kakigi et al., 2005; Lagopoulos et al., 2009) showed that meditation increased  $\alpha$ -2 activity whilst simultaneously decreasing the sensation of pain. Following this trend Babiloni et al (Babiloni et al., 2005) showed that  $\alpha$ -2 activity decreases in the contralateral hemisphere with the induction of a combined stimulus of pain and movement (Babiloni et al., 2005). Perception of pain, especially limb pain has been further localized to the dorsolateral prefrontal, the primary somatosensory, motor and supplementary motor cortex (Lorenz et al., 2003).

The earlier mentioned findings support the existence of a relationship between increased  $\alpha$ -2 activity in the contralateral premotor, motor and somatosensory cortex and the subsequent inhibited perception of pain 12 h post-EIMD induction (Egsgaard et al., 2009; Lorenz et al., 2003; Palva and Palva, 2007a; von et al., 2000). Therefore an increased  $\alpha$ -2 activity in our cohort might be responsible for the dissociation between the sensation of pain and the changes in neuromuscular function caused by the muscle damage. This is of clinical importance as pain during the first 12 hours after the induction of EIMD does not reflect on the amount of damage caused.

Therefore we propose that an increased contralateral fronto-central  $\alpha$ -2 activity acts as a cortical top down regulator of the perception of pain 12 h post-EIMD induction and therefore leads to the delayed –onset pain response associated with EIMD.

There was a visible increase in  $\alpha$ -2 activity in the control group at 132 h (Figure 4), but this was not significant. We suggest that these changes are a consequence of a learning or familiarization phenomenon, although we have no descriptive data to confirm this. As a result of repetitive testing sessions control participants probably had a lower attentiveness and

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increased movement automation (Zhuang et al., 1997). An increase in  $\alpha$  is associated with this lower attentiveness (Sauseng et al., 2006).

This novel study explored the relationship between the neuromuscular changes and pain induced by an EIMD protocol and associated changes in EEG  $\alpha$ -1 and  $\alpha$ -2 activity. However our study investigated changes in induced  $\alpha$  activity, rather than event related  $\alpha$  activity and therefore includes, pre movement, movement and post movement recordings. The aim was to investigate the influence of pain and changed neuromuscular function on  $\alpha$  activity during a movement task. Also our data were not baseline corrected but rather compared to pre-EIMD protocol values to identify differences in activity between the groups post- versus pre-EIMD. The authors acknowledge that other factors such as changes in pain pathways or inflammatory processes could have lead to the dissociated response of neuromuscular changes and the pain response, but the interest of this study was how EIMD and its associated symptoms affected the  $\alpha$ -1 and  $\alpha$ -2 activity measured over the cortical areas. Further research is needed to integrate not only the pain and neuromuscular response with the EEG recordings but also possible inflammatory changes and adaptations in the pain pathways. Further studies should consider correlations between EMG and EEG, as well as look at a broader spectrum of EEG frequencies (including beta, theta and gamma).

EIMD is an interesting model of acute pain as the neuromuscular function is significantly disturbed within the first 12 h after the EIMD protocol while the onset of pain is delayed and only peaks 36 to 48 h after the induction of EIMD (Plattner et al., 2011; Proske et al., 2004).

Therefore we propose that the increase in  $\alpha$ -1 activity, 12 h after the EIMD protocol, may be part of a neurocognitive top down regulator of neuromuscular function (Kakigi et al., 2005; Klimesch et al., 2007; Proske et al., 2004; Sauseng et al., 2005) and that  $\alpha$ -2 activity may be a top down regulator that suppresses the sensation of pain (Kakigi et al., 2005; Klimesch et al., 2007; Sauseng et al., 2005).



## **PERSPECTIVES**

Over decades changes in peripheral symptoms associated with EIMD have been documented. Structural changes, increases in serum creatine kinase, inflammatory processes, swelling and decreased neuromuscular functioning have been well documented but none have provided adequate explanations for the delayed pain response associated with EIMD (Plattner et al., 2011; Semmler et al., 2007) . Therefore the novel approach of this study was to explore the response of the central nervous system, especially the cortical  $\alpha$ -1 and  $\alpha$ -2 activities, peripheral symptoms and pain response to the unaccustomed exercise caused by the EIMD. There is surmounting evidence that these brain areas and frequencies may regulate the pain response acutely (12h) while experiencing the symptoms of EIMD during movement. Therefore future research should investigate further associations between EIMD, acute pain and neuromuscular responses linking EEG, EMG and fMRI measures to unravel the afferent and efferent interactions between the muscle and the brain during EIMD as well as acute pain. In the future, the information could add to the understanding and management of EIMD in athletes.

## **ACKNOWLEDGEMENTS**

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## **FUNDING**

This study was funded by the Medical Research Council of South Africa, Discovery Health and the University of Cape Town.

**ETHICAL APPROVAL**

The study was approved by the Human Ethics Committee of the Faculty of Health Science (REC REF: 090/2004), University of Cape Town according, while the principles outlined by the Declaration of Helsinki were adopted in this study (, 2002).

**CONFLICT OF INTEREST**

The authors declare that they have no conflict of interest

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## CHAPTER 9

### Conclusion and Future Directions

There is a lack of information about brain activity associated to motor behavior and exercise in the field of sports medicine. Sensorimotor control is suggested to be the physiological background behind motor behavior and is able to provide accuracy and adaptability in movement coordination to a changing environment which is important for performance enhancement as well as injury care and management. This work (Chapters 6-8) adds new knowledge in the area of sensorimotor control in sports and exercise:

- (1) It had been demonstrated that EEG measures are feasible in complex sports related real-world environments and conditions for examining changes in EEG power
- (2) It had been demonstrated that EEG power measures are sensitive to variations in perception and action circuits due to the working-memory-hypothesis in sensorimotor control in different conditions related to sports medicine research:
  - a. Stress-related conditions affect brain activity in terms of sensitivity of EEG power after different fatigue protocols. EEG power analysis is able to demonstrate variations in brain activity in frontal Theta power associated with attentional demands and parietal Alpha-2 power related to sensory information processing demands
  - b. Sports-related conditions demonstrate changes in brain activity related to skill level and different environments using the golf putt as a model. Alterations in frontal Theta power and Alpha-2 power are linked to attentional and information processing demands
  - c. Injury-related conditions cause alterations in brain activity due to injury and pain. Alterations are demonstrated due to variations in frontal Theta power and parietal Alpha-2 power connected to demands in attention and sensory processing.

The results provide evidence that the working memory plays a role in sensorimotor control in sports and exercise. The variations in demand can be measured by EEG and propose that frontal and parietal areas are involved in sensorimotor control and fit the working-memory-hypothesis. More studies – especially longitudinal studies - are needed to further clarify the dynamics of brain activity related to the working memory and its role in sensorimotor control to develop the foundations for training strategies and monitoring.

The knowledge about the involved brain areas is important, but only the starting point to elucidate neural mechanisms behind sensorimotor control. With the knowledge “where to look” we should be able to increase the “information density” about neural processes in sensorimotor control. One of the most interesting aspects of the working memory is that it connects perception and action circuits. This idea is based on the knowledge that neuronal networks communicate with each other. Even here, the EEG can provide a measure of electrophysiological communication which is termed connectivity (Jirsa & McIntosh, 2007). Connectivity can be divided into structural, functional and effective connectivity. Whereas structural connectivity describes the static anatomical structure of the brain and can be measured by MRI, functional and effective connectivity refer to coupled activity between two distinct neuroanatomical areas and can be obtained by fMRI and EEG (in the case of sensorimotor control frontal and parietal areas as demonstrated in Chapters 6-8). The advantage of effective connectivity is the fact that it is directed which means it describes a driver-receiver relationship. Thus, it will be a future challenge to increase the amount of information about the working memory in sensorimotor control by including EEG connectivity measures in real-world environments and sports-related conditions.

The technical possibilities in the field of EEG have emerged enormously in the last five years. These possibilities include high-density EEG hardware with new dry and active electrodes as well as sophisticated signal processing algorithms (e.g. ICA, Source Localization, Granger causality models; Delorme et al., 2011). With these advancements knowledge about the brain related to motor behavior and exercise will increase and add important data to the field of sports medicine. Thus, the last barrier in sports medicine research – the brain – can be



successfully challenged by research approaches and results can help to develop new neurophysiological based tools for testing human performance and to optimize training strategies for performance enhancement and injury management. A combination of these neurophysiological tools with the standard measures of cardiovascular and neuromuscular function will provide not only athletes and coaches, but from a health perspective all people with a holistic approach to increase performance in health and disease.

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